Henry Ford Hospital Medical Bulletin

Vol. 6 December, 1958 No. 4
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Address communications to: Chairman, Publications Committee, The Henry Ford Hospital, Detroit, Michigan

HENRY FORD HOSPITAL MEDICAL BULLETIN

Published quarterly by the Professional Staff of the
Henry Ford Hospital and The Edsel B. Ford Institute for Medical Research

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Vol. 6 December, 1958 No. 4
Part I

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Henry Ford Hospital
1958

SOME OBSERVATIONS ON BONE MINERAL IN A CASE OF VITAMIN D RESISTANT RICKETS

HAROLD M. FROST, M.D.*

ABSTRACT

Using a new sectioning technique, material from a case of proven vitamin D resistant rickets has been examined, stained and unstained, in the fresh state. Osteoid seams are rapidly and unequivocably evaluated and lined about 90% of all the cortical vascular channels. In addition, a mineralization defect was found surrounding about 25% of the osteocytes present. This is a newly described phenomenon, and it is suggested that it is evidence of a disturbance of osteocyte metabolism.

INTRODUCTION

Cases of vitamin D resistant rickets used to be classified as dwarfism, due to dyschondroplasia, due in turn to an hereditary defect. With the discovery of vitamin D[3,4,5] and the recognition of its importance in the formation of the skeleton in post-natal life came the gradual realization that D resistant dwarfs were an unusual form of rickets.[7] Then followed the discovery of incomplete tubular reabsorption of phosphate which in turn led to the discovery that D resistant rickets is a juvenile form of this condition, otherwise known as renal phosphatasia or phosphate diabetes.[1] Subsequent treatment with supplemental phosphate has markedly improved our ability to manage the condition.

The older literature particularly is replete with pathological descriptions of the gross and microscopic anatomical alterations in D resistant rickets.[6,7] Of late an increasing amount of histochemical information has been added to the established morphology. Little work has been done on the bone mineral in these cases, however, save for microradiography. The reason is two-fold: scarcity of biopsy material and lack of a suitable sectioning technique.

I have developed a sectioning technique which is rapid, economical, simple and allows preparation of perfectly fresh sections of bone without decalcification, embedding, dehydration, work heat or production of significant work trauma.[2] Examination of such material led to the present paper.

Since the features to be described will not be familiar to the average reader, the appearance of normal bone sections such as above will be described first and then the appearance of the sections from the case of D resistant rickets to be described. The reader should realize at the outset that the descriptions will be of features which are due to the presence or absence of mineral in the bone, rather than staining characteristics of the soft tissue, and that the appearance of fresh, undecalcified material is markedly different from that of the routine H&E stain on decalcified and embedded material.

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THE PATIENT

I. K. a 9 year old Caucasian male was first seen at Henry Ford Hospital in March, 1958. Bowing of both lower extremities had first been noted at age 8 months and had been present thereafter, being the reason for his admission to Henry Ford Hospital. The diagnosis of D resistant rickets was made from physical, X-ray and laboratory evidence at the age of 3 years and was subsequently confirmed at different times by a famous eastern and another famous midwestern medical center. He had been on large doses of vitamin D since age 4 years. The patient's mother and maternal grandfather both have markedly short, bowed lower extremities.

Physical examination revealed marked bowing of femurs, tibias and fibulas. There was minor bowing of the forearms. X-rays revealed widened, irregular epiphyseal lines, irregular thickening of the epiphyseal plates and the aforementioned bowing. Alkaline phosphatase was 16.4 (Bodansky), serum phosphorus 2.0 mg%, serum calcium 9.7 mg%. He had been taking between 50,000 and 100,000 units of vitamin D per day. Tubular phosphate reabsorption was 75% (90% plus is normal).

Osteotomies of both femurs and tibias were done to correct the severe bowing present. The bone removed at this time was reduced to multiple sections by my technique the same day and examined immediately or stained and then examined.

APPEARANCE OF NORMAL BONE

Unstained sections of normal adults from 10-30u thick and mounted either in water or in synthetic resin are quite transparent. With full illumination of the objectives used, there is little detail that can be made out in the bone mineral itself. Partial illumination (due to phase effects) and phase contrast reveal granularity of the various lamellar zones which is due mostly to the mineral present since little granularity remains after decalcification by drawing dilute acid under the cover slip. The osteocytes, canaliculae and Haversian canals are visible due to differences in refractive index between the bone and the mountant. Contrast is much better with water as a mountant than with the standard histological synthetic resins.

If the section be allowed to dry in air or on a warming table, air then fills the spaces of the osteocytes, canaliculae and vascular channels.

An excellent stained preparation can be made by staining the section 48 hours in 1% basic fuchsin, removing the surface stain and mounting. The consistent feature of this stain is that it does not penetrate the walls of the spaces mentioned above unless there is a lack of mineral in the matrix. Most normal adult bone therefore is fuchsin impermeable and the stain will be deposited as a thin layer on the walls of the spaces in the bone. New bone and bone which for one reason or another has lost some of its full complement of mineral will be diffusely permeated by the fuchsin. This interpretation has been proven recently in our laboratories by microradiography done by Mr. J. Parsons.

Where new bone or pathological bone which is fuchsin permeable occurs, there is a continuous spectrum of densities of staining, indicating variations in the amount of fuchsin permeability. However, the decrease from maximum to minimum is not

accompanied by localization of the last remnants around the osteocyte. There is almost no gradient, the stain density decreasing almost uniformly overall.

D RESISTANT BONE

Two features of D resistant bone will be described: the osteoid seams and the pericellular defect.

(A) On fresh, water-mounted sections osteoid seams are immediately apparent with the 16 mm. objective as glasslike, hyaline rings lining the walls of vascular spaces. Outboard of the seam where mineralization is in progress and partially completed considerable granularity exists, and the thicker the section the more marked is the contrast between the two. In the present case the seams line about 90% of 348 vascular spaces counted and varied from 10-60u in thickness. Under polarized light they were distincly less birefringent than the outlying bone even after decalcification, suggesting that there is a disturbance in collagen formation in the seams. If a section be observed while drying in air, shrinkage of the seam is seen after evaporation of the droplet of water in the Haversian canal. At room temperature the loss of width is 50%; at 40°C on the warming table for 10 minutes it shrinks 75-90% of the original width, retaining its glasslike feature all the time. The mineralized bone outboard of the seam shrinks only 2.2% on cross section, 0.4% on longitudinal with the above treatment.

Sections stained with basic fuchsin as mentioned previously reveal that in every case where an osteoid seam exists the bone immediately outboard of it is fuchsin permeable for a variable distance. The stain density is maximal at the junction of mineralized bone with seam, minimal at the periphery of the osteon. The seam itself takes a homogeneous, dense stain which is not qiute impenetrable optically. Destaining 48 hours in tap water differentiates the seam from bone because the former loses much more stain than does the latter.

(B) Unstained, water mounted sections of the biopsy material from the case of D resistant rickets described above reveal a curious dark, granular blur with poorly defined edges surrounding about 25% of 642 osteocytes counted at random. This is a unique phenomenon, not having been observed in the previous 4 years work with sections of this type made from over 100 different patients of all ages, nor in 20 different animals. The granularity under high numerical apertures is seen to be due to multiple fibrillae about 0.2 x 3u, spiralling in the presumed direction of the collagen bundles in the area.

Allowing the above sections to dry at room temperature under observation reveals that water in the blurred area evaporated from the matrix to be replaced by air. The blurred area (I have coined the term *halo volume* to describe the mineral immediately surrounding osteocytes) is visible with full or partial objective illumination, by phase contrast and on interference microscopy is of less optical density than the surround.

On fuchsin stained material the blur above is stained red. Of incidental interest is the stainability of this halo volume by CoS, Ag, Ag_2S, PbS, Alizarin Red S and $KMnO_4$ techniques developed by me.

DISCUSSION

While it is self-evident that one case does not encompass the total pathology, there can be little doubt that the appearances described are real and probably represent this disease state.

The purpose in describing the osteoid seams was to reveal how easily and unequivocably seams can be detected in fresh, undecalcified, undehydrated material. Since it takes about 3 minutes to make and mount such a section, the method offers considerable promise in clinical biopsy situations where an osteomalacic process is suspected. It is well known that the demonstrability of osteoid seams by the standard H & E decal techniques is uncertain. Minor variations in the total technique may alter the difference in staining sought for as a means of identification. There is on the other hand no uncertainty in the fresh, undecalcified material.

Numerous counts of seams in material from patients of all ages without an osteomalacic process reveal that in the healthy adult the incidence of seams is about 3 per 1000 osteons. In children the count ranges considerably higher, up to 100 per 1000. Accordingly, the 900 per 1000 present in I. K. is markedly elevated.

A final note of interest is the poor birefringence of the osteoid seams in their wet state. This is characteristic of seams in general, not just in the present case, and suggests very strongly that the seam is not normal matrix but is lacking in total collagen content or in the organization characteristic of collagen in normal bone matrix. This in turn suggests that the seam is the result of a metabolic disturbance in the cells forming it rather than the result (only) of mineral deficit.

The fuzzy blur around 25% of the osteocytes in the present case of D resistant rickets is intriguing. The microscopic observations leave no room for doubt about the reason: there is incomplete mineralization in the affected volume of bone. Since standard H & E decal sections made of the same case reveal some excessive basophilia but not lack of matrix in this volume, a quantitative lack of matrix is unlikely. A qualitative change is probable, however.

It is widely assumed that the mineralization defect in rickets is due solely to lack of mineral elements in the blood and that there is no primary metabolic disturbance in the osteoblasts or osteocytes. The pathological halo volumes present here can however be interpreted in the opposite manner. It is possible that the enzyme defect that makes the renal tubular cell unable to remove phosphate from the glomerular filtrate is more wide-spread and affects other cells including the osteocyte. It is certain that if a mineral deficiency alone were the cause of the mineralization defect in rickets the parts of the bone farthest from the cells, not closest to them, should be most affected, since passage of mineral from the canaliculae, through the cell and into matrix indicates that of necessity the concentration gradient must be positive, that is, maximal at the cell, minimal at a distance from it.

The mineralization gradient in fact is negative. This could be interpreted to mean that some metabolic product of the osteocyte in D resistant rickets *prevents* mineralization from occurring close to the cell where its concentration is maximal.

In other words, I suggest that there is a primary, cellular metabolic defect in this disease which involves the osteocyte. This defect may involve phosphate metabolism also but not necessarily so.

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Figure 1

Cross section ulna undecalcified, 20 yr. male, 700X, N. A. -0.95 Basic Fuchsin. Three osteocytes and parts of some of their canaliculae are in focus. Stain does not penetrate bone walls of physiologic spaces. The canaliculae are so small (0.4u diam.) that they are defined with difficulty.

Figure 2

Cross section humerus 20 yr. male. 1000 x, N.A. 1.32-undecalcified. HgS stain. Osteocyte lacuna and parts of canaliculae close to it are stained by virtue of HgS precipitated in bony wall of the spaces. Penetration of the wall about 1.0 u. CoS, Ag, Ag_2S, PbS, $Kmn0_4$ and Alizarin Red S produce similar stains.

Figure 3

Cross section tibia. I.K. (subject of text). 300 X, bright field, N.A. -0-65, 50% illuminated. Unstained, water mount. The osteoid seam is the hyaline ring surrounded by granular, partly mineralized bone. Section 30u thick. Contents vascular canal avulsed by section preparation.

Figure 4 A

Cross section tibia, I.K. unstained, undecalcified, 100X, bright field N.A. -0.65 reduced 50%. The fuzzy blur referred to in text can be seen affecting 5 osteocytes to a marked degree, 6 to a lesser degree. Normally mineralized bone never shows this phenomenon.

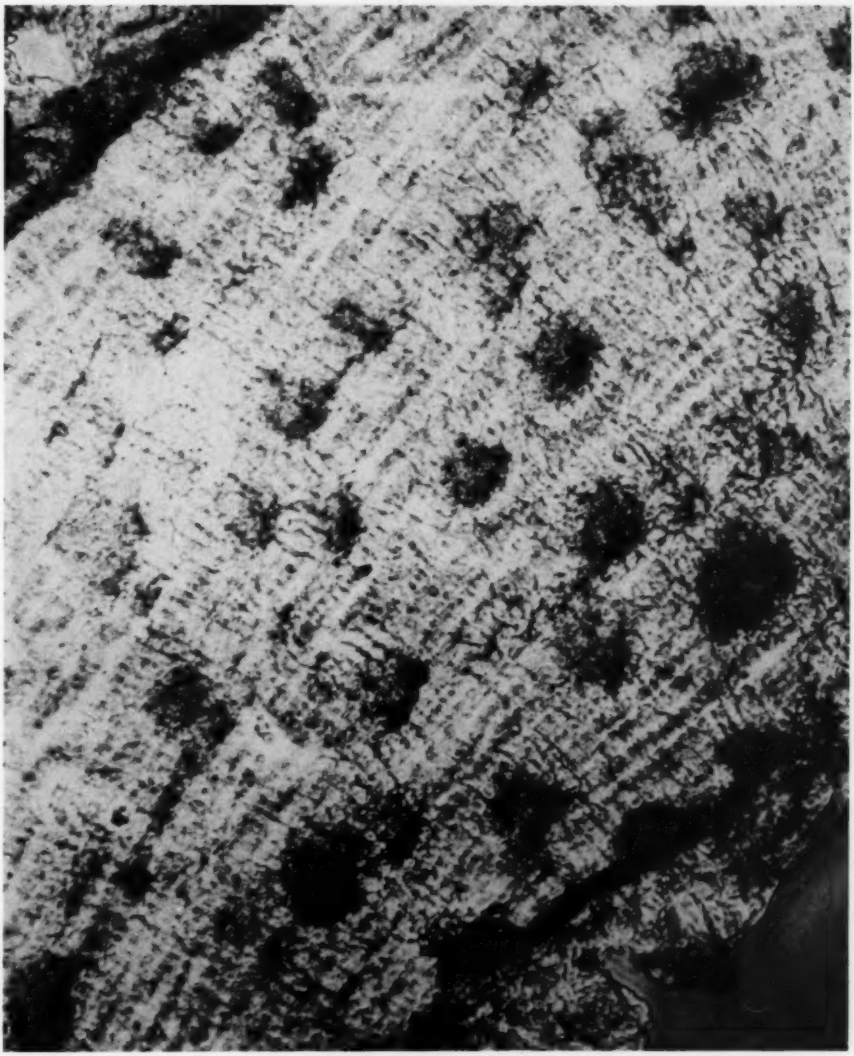

Figure 4 B

Longitudinal section opposite tibia same optical factors. Section dried at room temperature. The granularity in the blurred zones surrounding many of the osteocytes is more apparent due to accentuation of differences in refractive index by using air as a mountant.

Figure 5

Cross section, I.K. undecalcified, 1000 X, N.A. 1.32, full illum, stained with CoS. Compare with Figure 2. The cobalt has diffused widely beyond the confines of the two osteocyte lacunae in the centers which are blacked out. This is a pathological "halo volume".

PLATELET ABNORMALITIES AND PLATELET TRANSFUSIONS*

SHIRLEY A. JOHNSON, Ph.D.**

Certain platelet abnormalities may indicate a need for replacement of platelets by either platelet rich blood, platelet transfusion or platelet substitutes. When it has been established that a patient does need platelets to control bleeding, the actual form of this platelet replacement is a problem. It is only fair to say that most investigators probably feel that the ideal platelet form for transfusion has not yet been found. Platelet rich blood which is blood collected in plastic bags is perhaps the easiest, most effective form. Ordinary bank blood does not contain many platelets while fresh blood collected in a plastic bag is an excellent source of platelets. As the patient is usually bleeding, replacement of whole blood is a good idea. The difficulty with this is that often it is necessary to concentrate the platelets, for the necessary amount cannot be given in whole blood. Fresh intact platelets can be given, the platelets being collected and isolated in a plastic bag, but this takes time and the average blood bank would have difficulty supplying fresh platelets to all patients who need quantities of platelets, not to mention the economics of the situation. A platelet substitute that is inexpensive and easy to prepare is what is needed and that is what is difficult to develop. Lyophilized human platelets collected from every bottle of bank blood is perhaps the most popular approach.[1] It is true that these are platelets that in the average blood bank are wasted but on the other hand platelet factor 3 is largely destroyed by lyophilization. Also there are immunological problems to be considered, at least theoretically.

A substitute for platelets may be the answer. Soybean lipids have been studied and phospholipids in general have received attention in this investigation. Exactly what characteristics of a phospholipid render it effective in blood coagulation are not understood.[2] Some evidence suggests that phospholipids which have unsaturated fatty acids are the active ones and some other evidence points to the particle size of the phospholipid as the important characteristic.

We have attempted to purify bovine platelet factor 3[3] from bovine blood. Platelet factor 3 is undoubtedly a phospholipid and, in vitro, is an extremely active one. That is, a very small amount activates a great quantity of purified prothrombin. However, a large amount is needed to transfuse a patient who has a low platelet count in order to see a prolongation of the abnormally short prothrombin consumption time[4] of this thrombocytopenic blood. The fragility test, which is the result of the exertion of a given amount of suction on the skin, is improved always when purified platelet factor 3 is transfused. Many hemorrhagic areas appear before a transfusion is administered. After the transfusion, however, very few hemorrhagic areas result when the same amount of suction is applied. This means, that some protection is offered to the capillary wall by the transfused platelet factor 3 but rarely is an improvement in the prothrombin consumption time seen. It is possible that the platelet factor 3 is

*Presented before the Fourth Annual Meeting of the Michigan Association of Blood Banks, October 18, 1958.

**Department of Laboratories.

performing two functions. Perhaps it is acting on the capillary wall in a physical way and at a higher concentration it plays a role in the chemical reactions of blood coagulation.

Table 1 illustrates the role of platelets in coagulation as it now appears. There are 11 platelet factors listed here and there well might be more. Some of the platelet factors so far described appear to have activities similar to previously studied plasma factors. It is probable that platelet factor 3 is the most important platelet factor in blood coagulation. The importance of the platelet in coagulation must hinge on an activity not present in plasma; otherwise, why would blood coagulation be altered when platelet numbers are reduced.

Table I

PLATELET FACTORS

1. Platelet factor 1 or platelet Ac-globulin
2. Platelet factor 2 or fibrinoplastic factor
3. Platelet factor 3 or prothrombin activation factor
4. Platelet factor 4 or anti-heparin factor
5. Antifibrinolysin factor
6. Platelet co-thromboplastin or snake venom factor
7. Clottable factor
8. Anti-thromboplastin factor
9. Ac-globulin stability factor
10. Serotonin or vasocontriction factor
11. Clot retraction

Platelet factor 1 or the platelet Ac-globulin was first described by W. H. Seegers and his associates at Wayne State University.[5] Several years later it was shown that the blood of patients lacking Ac-globulin contained blood platelets which also lacked platelet Ac-globulin.[6] These same patients' platelets after suspension in normal plasma picked up Ac-globulin activity presumably from the plasma. In spite of some kinetic difference between preparations of plasma Ac-globulin and platelet Ac-globulin[5] this experiment indicated that at least Ac-globulin could be picked up from the plasma.

There is considerable evidence that the platelet antifibrinolysin, factor number 5 on the table,[7] is different from plasma antifibrinolysin according to kinetic studies of Alkjaersig and Sherry.[8]

The clottable factor or platelet fibrinogen may or may not be different from plasma fibrinogen. Blomback has kinetic studies indicating that they are the same, while a patient of Dr. Monto's was found to have a normal plasma fibrinogen level and about one third of the normal platelet fibrinogen which is evidence that the two are different.[9]

To be sure that two proteins are different requires exhaustive chemical characterization carried out on highly purified preparations of the proteins in question. In each case involving these platelet factors such highly purified preparations have not yet been available.

It appears then that platelet factor 3 is the only known platelet activity effective in coagulation that is not found in plasma. It could therefore give to the platelets the characteristic qualities required in normal hemostasis. This indicates that on a theoretical basis and a practical one as well, replacement of the patients' platelets is in fact replacement of platelet factor 3. There appear at the moment to be three clinical situations in which platelet factor 3 is unavailable to the patient and needs to be replaced, thrombocytopenia,[10] congenital thrombocytopathy A,[11] and acquired thrombocytopathy of uremia.[12]

Thrombocytopenia will be dealt with first because an assay procedure measuring platelet factor 3 activity has been used involving thrombocytopenic blood. We have defined the thrombocytopenic state as that involving a platelet count of 50,000 or less. It was first reported by M. Stefanini, that blood in the thrombocytopenic state gave poor prothrombin consumption time.[13] This was the first and is still one of the best pieces of evidence that platelets are involved in the chemical mechanisms of coagulation. Some investigators believe that platelets act only in a physical manner such as plugging up the holes in the capillary bed. Other investigators believe that platelets actually enter into the chemical reactions of blood coagulation. A normal prothrombin consumption time is 30 seconds or more, an abnormal one 20 seconds or less and 20 to 30 seconds is borderline.[4] A preparation of platelets or platelet factor 3 can be assayed by adding it to blood in the thrombocytopenic state and observing the corrective effect on the prothrombin time. That is, a short prothrombin time of 15 seconds can be prolonged to 35 seconds or more. The prothrombin consumption time is a prothrombin time on serum and is consequently prolonged when prothrombin and its related components disappear as blood clots and serum is formed.

In addition to this method of assay, we have a more quantitative one. Platelet factor 3 with the antihemophilic factor will activate purified prothrombin to thrombin quantitatively.[3] It is possible to replace the antihemophilic factor with some of the antihistamines, for example, Benadryl. For assay purposes it is much more reproducible to use a synthetic compound such as Benadryl rather than a labile protein such as the antihemophilic globulin. In general, we have found that both assay procedures correspond well.

The blood in the thrombocytopenic state gives a prothrombin consumption time of 20 seconds or less.[10] This serum, however, does not contain residual prothrombin in the way that hemophilic serum does. Usually hemophilic serum contains about 180 units per milliliter of prothrombin, normal plasma about 230 u/ml and normal serum 20 u/ml. Therefore, it seems logical to assume that the short prothrombin consumption time of hemophilic serum is due to residual prothrombin. The short prothrombin consumption time of blood in the thombocytopenic state is due not to prothrombin but to the formation of some accelerator formed when blood clots, for there are less than 40 u/ml prothrombin in the serum. This accelerator appears

chemically to be very much like prothrombin, in that it is readily adsorbed on inorganic chemicals, but does not possess prothrombin activity according to the two-stage prothrombin time.[10] Extensive purification studies on this accelerator have been carried out and the method of purification used is outlined. Thrombocytopenic serum is concentrated in the ultra-centrifuge by centrifugation for 18 hours at 94,000 g. The activity does not appear to change but the protein concentration is greatly increased as can be seen in the Tyrosine determination which is increased from 2.88 mg/ml to 4.72 mg/ml. The bottom layer of serum which contains the activity can be precipitated with 50% saturation of ammonium sulfate leaving the activity in the supernatant. After dialysis the activity can be adsorbed on Kaolin and eluted with phosphate buffer. The second eluate contains the activity and very little protein indicated by a Tyrosine value of 0.078 mg/ml.

This basic pattern of platelet factor 3 deficiency in the thrombocytopenic state, which has been described, was found in the two types of thrombocytopathy, the congenital type and the acquired thrombocytopathy of uremia and can be seen on Table II.

Table II

	Hemophilia	Thrombocytopenic State	Thrombocytopathy A Congenital	Thrombocytopathy of Uremia Acquired
Platelet count	mm^3 Normal	mm^3 50,000 or less	mm^3 Normal	mm^3 Normal
Prothrombin Consumption	Under 20 seconds	Under 20 seconds	Under 20 seconds	Under 20 seconds
Prothrombin Consumption $BaCO_3$ adsorbed serum	Over 50 seconds	Over 50 seconds	Over 50 seconds	Over 50 seconds
Residual Prothrombin in Serum	180 u/ml	Under 40 u/ml	Under 40 u/ml	Under 40 u/ml

In congenital thrombocytopathy the patients have a bleeding difficulty in the face of normal platelet numbers and a poor prothrombin consumption time with no other coagulation difficulty.[11] When the platelets from these patients are collected from 100 ml. of blood and separated by differential centrifugation, it can be observed that they are deficient in platelet factor 3 activity. Normal platelet extract activated nearly 800 units per milliliter purified prothrombin to thrombin while the patients' platelets in each of the cases studied activited less than 100 units per milliliter.

Dr. Rebuck has found that the platelets of these patients suffering with congenital thrombocytopathy were abnormal morphologically. It is believed that platelets undergo certain changes as disintegration takes place.[11] The postulated phases are round, dendritic, intermediate and spread. When differential counts of 100 platelets were

carried out using the electron microscope, it was found that most of the normal platelets were in the dendritic state and most of the patients' platelets were in the last two stages of disintegration, intermediate and spread. (Table III)

Table III

ELECTRON MICROSCOPE PLATELET DIFFERENTIAL COUNTS IN A TYPICAL CASE

	Round	Dendritic	Intermediate	Spread
Normal	0	82	8	10
Thrombocytopathy A	1	29	51	19

It appeared from this that the patients' platelets may have had difficulty in disintegration, and so the platelet factor 3 activity was measured after the platelets had been disintegrated mechanically in the sonic oscillator. It was found that the platelets of these patients contained as much platelet factor 3 activity, when thoroughly liberated, as normal platelets.

The similarity of thrombocytopathy to thrombocytopenia becomes apparent for it can be seen that thrombocytopenia is an absence of platelets and therefore of platelet factor 3. Thrombocytopathy is a lack of available platelet factor 3 in the presence of adequate numbers of platelets. In the coagulation mechanism the similarity is also apparent. Blood from both the thrombocytopenic state and thrombocytopathy result in a poor prothrombin consumption time with little or no residual prothrombin in the serum. That is, the short prothrombin consumption time in each case cannot be due to prothrombin left in the serum after the blood had clotted.

The third condition in which bleeding occurs due to an involvement of platelet factor 3 is in uremia.[12] Dr. Monto selected a group of severe uremic patients who had an impaired prothrombin consumption time. The platelets were collected again by differential centrifugation from a quantity of blood and the thromboplastin generation of these platelets was found to be less than that of an equal number of similarly treated normal platelets. It is interesting that the difference seen between normal and uremic platelets can only be observed when the platelet numbers are limited. Apparently each platelet has a limited amount of platelet factor 3 activity. There appears to be little difference in the thromboplastin generated by 200,000 mm^3 normal platelets and 200,000 mm^3 uremic platelets but a very significant difference between the thromboplastin generated between 100,000 mm^3 normal platelets and 100,000 mm^3 uremic platelets. The thromboplastin generation of plasma and serum of the patients was, of course, normal.

When tested in the platelet factor 3 assay, the uremic platelets contained very little platelet factor 3 compared to normal platelets. Disintegration of platelets by the sonic oscillator did not result in increase of this imparied activity. These platelets were either produced by abnormal megakaryocytes with each platelet deficient in platelet factor 3 activity or, they were formed possessing the activity which was then

destroyed by the abnormal metabolites or metabolites produced in abnormal amounts in the uremic blood.

It was possible to incubate normal platelet extract in uremic plasma and to consequently destroy the platelet factor 3 activity. After eighteen hours incubation at 8° C the platelet extract would only activate 200 u/ml of purified prothrombin to thrombin while the same platelet extract suspended in normal plasma activited 800 units per milliliter. Whatever the component is that rendered the platelet factor 3 inactive, we know it is contained in the uremic plasma and it destroys, not renders unavailable, the activity.

Again, as in the thrombocytopenic state and congenital thrombocytopathy, the poor prothrombin consumption time of uremic blood is not due to residual prothrombin in the serum but due to the formation of some accelerator as the blood clots. Any blood giving a poor prothrombin consumption time but with little or no residual prothrombin in the serum is probably blood with defective platelet function involving platelet factor 3.

There are three clinical conditions in which the mechanism of blood coagulation appears to be similar. Table IV summarizes the studies on the platelets of the three clinical states: thrombocytopenia, where there is an absence of platelets and consequently of platelet factor 3, congenital thrombocytopathy in which the platelets appear to possess the activity but appear to be unable to render the activity available and the acquired thrombocytopathy of uremia in which the platelets appear to lack the activity, which has been destroyed in some way.

Table IV

	Hemophilia	Thrombocytopenic State	Thrombocytopathy A Congenital	Thrombocytopathy of Uremia Acquired
Thromboplastin generation of platelets	Normal	—	Decreased	Decreased
Platelet Factor 3 Activity	Normal	—	Decreased	Decreased
After sonic treatment	Normal	—	Normal	Decreased

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THE RESULTS OF SURGERY FOR BRONCHIECTASIS

GEOFFREY L. BRINKMAN, M.D.* AND HANNES PAULI, M.D.*

The past 20 years have seen a great change in bronchiectasis, both in its clinical picture and in its treatment. The more frequent use of bronchoscopy and bronchograms has resulted in diagnosis at a much earlier stage, while the introduction of antibiotics has greatly improved the medical management. It is timely therefore to review the results of resection in order to better define those cases which are most suitable for radical therapy.

All cases of proven bronchiectasis seen at this hospital between 1946 through 1955 were reviewed. There were 110 such cases of which 53 underwent resection. The remaining 57 patients were unsuitable for surgery, either because of their age or because of the extent of their disease. Most of these non-operated patients have been lost to follow-up, but of the surgically treated patients, 100% follow-up has been obtained 1 to 11 years postoperatively. There were 13 males and 40 females whose ages range from 18 to 55, with the exception of one 10 year old girl.

RESULTS

Nineteen of the patients had bilateral disease but in only 2 patients was bilateral resection undertaken. Neither was benefited. One continues with as much cough and sputum as preoperatively, while the other is a respiratory cripple and is in congestive heart failure.

Although there were no immediate postoperative deaths, 2 patients have died. One death was due to infectious hepatitis 4 years postoperatively and the other occurred 2 years after lobectomy in a patient with bilateral disease who had recurrent infections and died in congestive failure.

Lobectomy or multiple segmental resection was the usual procedure, but pneumonectomy was carried out on 3 occasions. Two of these patients have done well but the third died of hepatitis as noted above.

Table I

Degree of Improvement	No.	%	
Excellent	21	40)	
		)	66%
Moderate	14	26)	
Unchanged or dead	18	34	
	53	100	

The present condition of those bronchiectatic patients treated by lung resection at Henry Ford Hospital from 1946 through 1955.

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Those individuals who were completely relieved of their symptoms were regarded as having an excellent result, while those who were significantly benefited but continued to have some daily cough and sputum were regarded as moderately improved. Using these criteria, follow-up examination, as reported in Table I, shows that 35 (66%) of the patients were improved. Nineteen patients with bilateral disease had unilateral resection, and only 8 (42%) of these patients were benefited (Table II).

Table II

Degree of Improvement	No. of Patients	%
Excellent	1	5
Moderate	7	37
Unchanged	11	58

The results of unilateral resection in patients with bilateral bronchiectasis.

DISCUSSION

Failure to improve after resection is usually due to one or more of the following factors:

1) Inadequate preoperative selection of patients.

a) Pulmonary insufficiency.

Thirteen of our patients complained of increased shortness of breath following operation; none showed improved exercise tolerance. In view of this, it is unwise to undertake resection if the patient already has significant pulmonary disability. The only exception to this generalization is when the circulation is maintained through a non-ventilating bronchiectatic lobe, resulting in a physiological venous-arterial shunt. Resection in this case will remove the shunt, and the arterial oxygen saturation will improve with consequent benefit to the patient.

b) Severe non-pulmonary disease.

The patient's general health may mitigate against operation. Two specific conditions which may present with the clinical picture of bronchiectasis are agammaglobulinemia and fibrocystic disease of the pancreas. Both are common in childhood but occasionally present in young adults, and so should always be considered in the differential diagnosis. Either condition would be an absolute contraindication to surgery.

c) Age.

Postoperative cooperation is difficult to obtain in children, so that retention of secretions with resulting atelectasis is more common. Such postoperative complications have a deleterious effect. At the other end of the lifespan, cooperation may be equally unsatisfactory, and combined with the degenerative processes of aging, this will adversely effect the results, as was demonstrated by Gudbjerg.[4]

d) Bronchitis.

Clinically, the diagnosis of bronchitis depends on hearing wheezes or crepitant rales in non-bronchiectatic areas of the lung. A more accurate method of making

the diagnosis is by bronchoscopic examination of the mucosa. Unfortunately, many of our patients did not have bronchoscopy at the time of their preoperative bronchograms, but in those that did, bronchitis was recognized in 8 out of 19 patients. Three of these 8 patients were not improved by surgery, even though their bronchiectasis was totally removed.

Helm and Thompson[7] also found bronchitis had an adverse effect. Of 32 patients diagnosed as having bronchitis preoperatively, none was improved by resection. Postoperative bronchograms in these same patients showed that 48% had developed further bronchiectasis.

e) Bilateral disease.

Incomplete removal of bronchiectasis cannot be expected to result in cure. It often fails even to achieve moderate symptomatic relief. In our own series, 56% of the patients were not benefited following partial removal of their bronchiectasis. Gudbjerg had a much higher mortality in this group with 13 of 32 such patients dying during a 5 year observation period. Seventeen of the 19 survivors developed further bronchiectasis.

2) Postoperative complications.

Ginsberg[3] showed that in those patients with an unsatisfactory end result, postoperative complications occurred twice as frequently, and in these patients, retained secretion was the commonest and most serious single factor. In the Henry Ford Hospital series, of the 7 patients with unilateral disease who did not improve, 4 had a postoperative bronchopleural fistula, and in 3 of these empyema developed necessitating rib resection and drainage. Gudbjerg's experience also confirms that a poor result often follows postoperative atelectasis or empyema.

3) Recurrence of bronchiectasis.

Only 3 of our patients have had postoperative bronchograms and these were normal. Chesterman[8] reviewed 273 patients who underwent lobectomy for bronchiectasis and found a recurrence rate of 9%, which he attributed mainly to postoperative atelectasis or infection. Twenty-four of 221 cases reported by Ginsberg had new bronchiectasis demonstrated postoperatively and in 62.5% of these postoperative complications had occurred.

In Helm and Thompson's series[7], 100 patients had postoperative bronchograms. Thirteen showed new bronchiectasis on the operated side, while a further 13 showed an extension of the bronchiectasis on the contralateral side.

Although resection remains the only curative therapy available, it is apparent from this discussion that the results cannot be guaranteed beforehand. There are now numerous reports in the literature (see Table III) which substantiate the unpredictable results of surgery.

CONCLUSIONS

Twenty years ago Perry and King[9] reported that almost half of their patients with bronchiectasis died within 5 years of the onset of symptoms. About the same time,

Table III

Author	No. of Patients	% Improved
Lindskog	129	69
Ochsner	82	67
Ginsberg	213	76
Paterson	118	50
Strang (children)	163	76
Gudbjerg	52	50
Helm & Thompson	159	61

The results of resection for bronchiectasis in several recently reported series (see References 1-7).

Bradshaw, Putney and Clerf[10] reported the average duration of life after diagnosis was less than 2 years. In the past, chest infection and its complications were the major cause of death in these patients, but now the skillful use of antibiotics, along with postural drainage, will maintain many of these patients in satisfactory health. Wynn-Williams'[11] experience with 166 bronchiectatic patients clearly illustrates this change in prognosis. Over 3 years of observation, only 6 of the patients died, and while 3 of these deaths may be attributed to bronchiectasis, none died of infection or its complications. Scannell[12] had a similar experience with 19 patients at the Massachusetts General Hospital who refused surgery and were therefore treated medically. None died of sepsis, although 4 died of massive pulmonary hemorrhage.

There is no doubt that bronchiectasis is now frequently diagnosed at a much earlier stage than it was 20 years ago, and the poor prognosis reported previously is no longer applicable. Medical therapy alone may adequately control many of these patients, and there is not the same urgency to resort to surgery.

SUMMARY

Postoperative results of 53 patients with bronchiectasis are reported. Approximately one-third of these patients were not benefited by lung resection. The reasons for this are discussed.

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CARDIAC SURGERY AND CORONARY ARTERY DISEASE

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The problem of angina pectoris has been managed most extensively by non-surgical means, but for many years cardiac surgeons have been attempting to relieve the pain of angina by methods which have in common the increase of blood supply to the myocardium. Though this approach to the problem is currently receiving much attention, it has been the subject of considerable investigation for at least twenty years.

The essence of the operations for angina pectoris is to increase the supply of oxygenated blood available to the myocardium. It has been found that as the arteriosclerotic process renders the coronary vessels less capable of providing the necessary blood to the heart, there is a gradual development of collateral circulation between the major vessels. Thus a fairly even distribution of blood is maintained. However, a certain critical level of muscle anoxemia may finally occur despite the collateral circulation, and, it is assumed, the angina develops when demands are placed upon the heart which are in excess of the adequacy of the coronary circulation. At this point the surgical approach to the disease seems most appropriate especially with reference to the patient who has gained maximum benefit from medical management.

The procedure which has been in use for the greatest number of years is poudrage which consists of introducing foreign material such as asbestos into the pericardial sac. This produces an initial inflammatory reaction which persists for years, the formation of granulation tissue, adhesion of the pericardium and epicardium, and eventual organized vascularization of the granulation. The procedure has been done by Thompson, and one of his patients is now eighteen years post-operative. A review of 250 of these patients as of 1957 is outlined as follows:[1]

No. Patients	Operative Death	Poor Results Less Than 50 % Improvement	Good Results At Least 50%	Excellent Results More Than 75% Improvement
250	7%	10%	50%	40%

By these figures approximately 90% of the patients had good to excellent results, and 10% were considered to have poor results. The operative death rate of seven per cent may seem excessive, but it must be remembered that many of these people would have been classified as poor risks for any type of procedure. Also it may be assumed that many of the poor result patients had such extensive damage to the myocardium that revascularization could not possibly have been of value.

The group headed by Beck[2] has worked extensively on revascularization procedures involving not only the poudrage but also partial ligation of the coronary sinus,

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pericardial and epicardial abrasion, and placing of a graft between the aorta and coronary sinus. The grafting technique has been abandoned, but partial ligation of the coronary sinus apparently produces a venostasis and anoxemia within the myocardium which serve as a stimulus to development of any collateral circulation available such as through the granulation tissue caused by the poudrage and abrasion. In a report by Brofman[2] published in March 1957, concerning 225 of these patients, there was an overall operative death of five per cent. However, Beck has recently had a series of over 100 patients who underwent the operation without a mortality. For the evaluation of 100 consecutive patients operated by Beck,[2] reference is made to the following chart:

No. Patients	Operative Death	Poor Results	Less Pain	Free of Pain	Return to Work	Pre-operative Incapacitation
100	5%	10%	45%	45%	90%	45% were economically productive

Again it is seen that ten per cent of the patients had poor results, and ninety percent were improved which is quite in accord with the series by Thompson.[1] Also it is to be noted that prior to the operation forty-five per cent of these patients were economically productive whereas post-operatively ninety per cent were able to work either full or part time.

A third type of procedure also has the purpose of increasing the blood supply to the myocardium, but there is a departure from the poudrage type of operation. Vineberg and Walker[3] have been working with implantation of the left internal mammary artery in the myocardium. When this vessel is secured in an area not adequately supplied with blood, new vessels will develop forming an internal mammary-coronary artery circulation. Vineberg and Walker[3] have a series of eighty-eight patients followed six months to five and one-half years, and these results are presented in the following outline:

No. Patients	Operative Death	Poor Results	Less Pain, Slight Pain, Pain Free	Work Post-operative	Incapacitation Pre-operative
68 no angina at rest	6%	18%	82%	84%	72% of total
20 angina decubitus	45%	37%	63%	54%	100%

In the larger group which contains the better risk patients the operative mortality was six per cent, but in the group with angina decubitus the mortality was forty-five per cent. Of the patients surviving in the large group, eighty-two per cent had less pain or were free of pain, and eighty-four per cent of this group was able to work post-operatively whereas the pre-operative incapacitation was seventy-two per cent of the total. Of the angina decubitus group only eleven survived with seven having less pain and six returning to work. Admittedly this is a rather small group from which

only an impression can be obtained, but some of the patients who were improved had previously been taking about 300 nitroglycerine tablets per week.

The fourth major procedure about which there is a great deal of controversy is that involving the internal mammary artery ligation. The rationale of the procedure is that the transection of this vessel causes blood to be shunted through the pericardiophrenic artery which anastomoses with vessels of the coronary circulation thus providing a collateral circulation to areas deprived of their primary blood supply. Injection studies of the internal mammary artery have demonstrated these anastomoses, and the occlusion of a coronary vessel of dogs before and after the procedure has shown that some protection from infarction is afforded the operated animals.

The group in this country which has done the most work along this line is headed by Glover.[4] They operated on any patients with the diagnosis of angina pectoris, and follow-up statistics for fifty of these patients are presented:

No. Patients	Operative Death	Poor Results	Less Pain	No Pain	Post-operative Work Capacity	Pre-operative Incapacitation
50		22%	32%	36%		

From these figures it can be interpreted that sixty-eight percent of the patients were improved and twenty-two percent were not benefited. No mention is made either of the post-operative functional capacity or of the death rate of the operation. It might be mentioned that since the publication of the above figures another fifty patients have been added to the series and the percentages are essentially unchanged.

There is a great deal of skepticism among internists concerning the efficacy of surgery in coronary artery disease, and in order to more fully compare results and to further evaluate the natural history of angina in regard to pain and functional capacity about 200 charts of patients diagnosed as having arteriosclerotic heart disease were reviewed by the author and sixty were selected. The basis of selection was a follow-up of at least one year at this hospital and the absence of a disease entity which obviously would cause death within a short time. The severity of the arteriosclerotic heart disease was graded as follows: zero — no pain; 1 plus — pain on moderate exertion, working as usual; 2 plus — pain on routine activity, able to do light work; 3 plus — pain on minor exertion, unable to work; 4 plus — status anginosus. The results of this investigation are shown in the following chart:

DEGREE OF ANGINA AT TIME OF DIAGNOSIS

	0	1+	2+	3+
Number of Patients	9	38	12	1
Angina Increased	56% (5)	47% (18)	42% (5)	
Angina Unchanged	33% (3)	37% (14)	50% (6)	(1)
Angina Decreased	11% (1)	16% (6)	8% (1)	
Average Year of Symptom Increase	2.4	3.4	4.7	

In this series the majority of the patients did not improve, and in the groups with the least symptoms initially there was a tendency to have increase in discomfort and incapacitation rather than to maintain a static state. It is interesting to note the increased number of years in the three groups before the pain becomes more acute.

The comparison of this limited group with the series compiled by the cardiac surgeons is destined for criticism, but some rather intriguing suggestions and observations can be made. In averaging the percentages of improved and unimproved patients operated by Thompson,[1] Beck,[2] and Vineberg,[3] it is found that eighty-seven percent are improved and thirteen percent are not improved. If in our group of sixty patients the unimproved and improved are averaged, the percentages are eighty-seven and thirteen respectively. The following chart more clearly presents the comparison:

	Surgical	Non-surgical
Improved	87%	13%
Not Improved	13%	87%

The results are rather striking which is often the case when a small group is used, but it is impressive that there is complete reversal of the percentages. It should be mentioned that all of the medical cases were surgical candidates in regard to the degree of angina, and to that extent they may be considered comparable. It would seem that the surgical approach has achieved a somewhat better relief of pain.

The question of longevity is pertinent to this discussion. There has been a surgical mortality of about five percent, but it should be re-emphasized that initially many of the patients were extremely poor risks. Beck[5] in reviewing a group of 137 consecutive cases followed six months to five years (average of two years) expected a thirty percent mortality in an unoperated comparable group, but actually there was only thirteen percent mortality. The patients in the Thompson[6] series have had an average life span post-operatively of five years with an average pre-operative symptomatology extending four and one-half years. Thus, the average life span is nine and one-half years which is comparable to a non-operated group. Again it must be mentioned that he was dealing with severely incapacitated patients the majority of whom had suffered one or more occlusions.

Thus, the controversy continues regarding the place of cardiac surgery in the management of angina pectoris, but evidence appears to be mounting in favor of surgical intervention in select cases. The present mortality at surgery is no greater than for any major procedure in the age group which would be most involved. Medical management is basically symptomatic and at present unchanging. Though the natural history of angina is variable, the majority of patients do not improve, and surgical intervention offers a ninety percent chance of improvement. The time has come when a study of angina from a functional capacity standpoint is indicated, and this should encompass two groups, the operated and unoperated. The time of operation could be arbitrarily established, perhaps at the point when the patient cannot adequately perform the work required. As always, there will be variables with which to contend, but by this approach the surgical management of coronary artery disease could be

evaluated with minimum risk to the patient. If the present operative statistics can be proved, the place of surgery will be definite, and the results will certainly be rewarding.

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THE USE OF JEJUNAL AND ILEAL LOOPS IN STOMACH AND COLON SURGERY

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The removal of all or part of the stomach and colon is a routine surgical procedure. All too frequently this surgery will remove the disease and the patient will be presented with new symptoms which are often more annoying or disabling than the original illness. So far, no plastic or substitute material from outside the body has been discovered to replace the stomach or colon.

In stomach surgery, all studies have strongly emphasized the protection of the gastric reservior against the development of cramps, diarrhea, weight loss, anorexia, etc. Small sections of jejunum have been used to make up for part of this loss. We have modified these segments of jejunum by the use of loops in the shape of a figure 9. Previously only short straight segments have been used. Figures 1 and 2.

A certain percentage of patients following partial gastrectomy get relief from the ulcer but go on and develop the distressing and disabling postgastrectomy syndrome ("dumping"). This is characterized by diarrhea, cramps, sweating, anorexiai, weight loss, weakness, etc. In these we have had excellent results by the transposition of a segment of jejunum as shown in Figure 2. In all cases of this type, we have used this procedure only where there was no free acid. Figure 3.

In total colectomy in the past, one has been forced to have a bowel opening on the abdomen. Colectomy, in which the major part of the colon is removed, has always been accepted with great reluctance because of the dread of going through life with an artificial bowel opening on the abdomen. At present there is great interest in trying to save the anus and lower rectum in certain cases of ulcerative colitis and multiple polyposis. The lower end of the ileum is then anastomosed to the rectal stump. The usual procedure in the past has been to anastomose the end of the ileum to the short rectal stump which is not covered with peritoneum or to close the end of the ileum and then attach the side of the ileum to the rectal stump. This gives a safer anastomosis but leaves a blind pocket which is most undesirable in bowel surgery. We have devised a method of anastomosis as shown in Figure 4. In this method we have a large peritoneal surface on the ileum to suture to the rectum. This gives a much safer anastomosis. Next we increase the storage capacity of the lower ileum. No blind loop is left in the bowel. We have been performing this with increasing frequency over the past several years. Figure 5.

CONCLUSION

The jejunal and ileal loops have proven to be of great benefit in selected stomach and colon cases. Our modifications have been presented.

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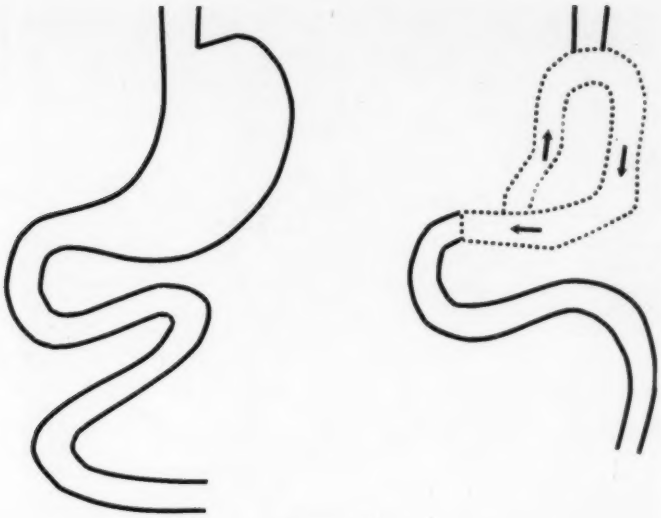

Figure 1

This diagram indicates that the entire stomach has been removed. A loop of jejunum with its own blood and nerve supply has been inserted. The arrows indicate the direction of the peristaltic waves. By this figure 9 loop of bowel, a large segment 14″ to 16″ can be inserted with no kinking. This gives ample storage space and is easily attached to the duodenum without tension. A large surface of bowel covered with peritoneum is available to anastomose to the esophagus which lacks peritoneum.

Figure 2

X-ray of figure 9 loop of jejunum inserted after total gastrectomy. Note the marked dilatation and ample storage space. This makes it possible for the patient to eat without discomfort.

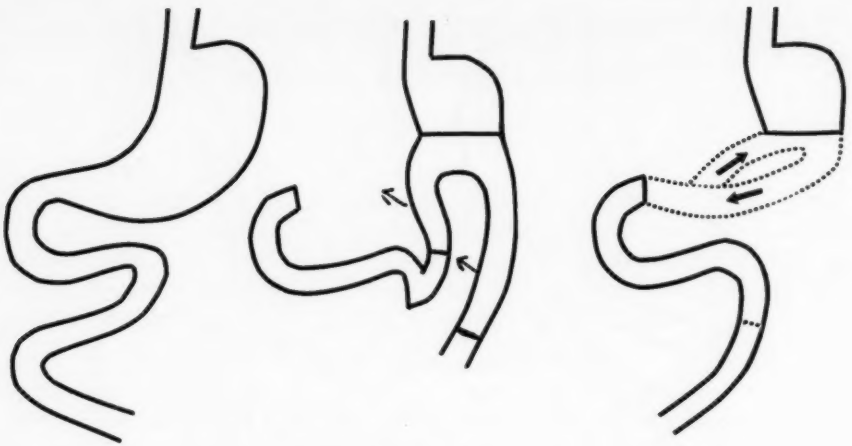

Figure 3

The proximal and distal jejunum are divided as shown. The original gastrojejunostomy does not have to be taken down and this makes the technical part of the operation much easier. The arrows indicate the peristaltic wave direction. The jejunal continuity is restored and the figure 9 loop is anastomosed as shown with the bottom of the 9 being attached to the duodenum. No blind loops are present.

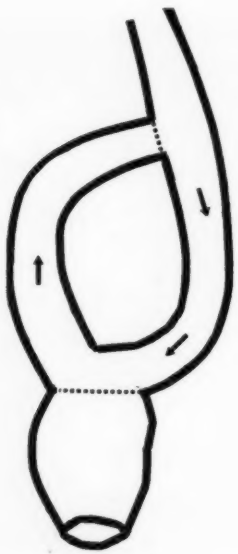

Figure 4

A figure 6 loop of ileum is anastomosed to the rectal stump as shown. A large, strong anastomosis is made by attaching the bottom of the loop 6 to the rectal stump.

Figure 5

X-ray of figure 6 loop ileal anastomosis to rectum at the level of the second valve. Note the great dilatation of the terminal ileum and the rectal stump.

RUBEOLA ENCEPHALITIS WITH HEMIPLEGIA; REPORT OF TWO CASES TREATED WITH CORTISONE

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AND WILLIAM C. MONTGOMERY, M.D.*

Based on experimental studies by Kabat[1] showing that the demyelinating process in animal encephalitis can be suppressed with Cortisone, steroid therapy has been used by clinicians in the treatment of measles encephalitis. Prior to this, it had been shown that gamma globulin was ineffective in treatment.[2] Allen, using steroids had complete recovery without sequelae in all 10 of his cases.[3] Appelbaum and Abler treated 17 consecutive cases with ACTH with no deaths; there was one case with neurological sequelae and one case of mental retardation and speech impediment.[4] Most of their cases were treated one week or less with a total of 100 to 300 mg. of ACTH. Unfortunately there was no control in either series, but both authors were impressed with the results in the light of their previous experience.

In neither of the above series was there an instance of hemiplegia. Tyler[5], in reviewing the literature on the neurological complications of rubeola, found 40 cases of hemiplegia including 10 of his own. Instead of a demyelinating process, many authors including Ford,[6] feel that the hemiplegias are probably the result of a non-specific effect on the vascular channels, such as one finds in many febrile illnesses. This being the case, the part that steroids would play is difficult to ascertain since the basic pathology is considered more likely to be infarction than perivascular demyelination. However, the exact mechanism of the hemiplegia is still open to question.

The most striking data found in Tyler's series were the persistence and the permanent residual of the hemiplegia, the abnormal electroencephalogram and high incidence of seizures. Of the 40 cases of hemiplegia he reported, 33 had persistent paralysis without recovery. None of the hemiplegia cases was treated with steroids.

Two cases of acute hemiplegia associated with rubeola are presented. Both of these cases had complete clinical recovery with no apparent residual. The remarkable reversal of symptoms in the cases reported would lead one away from the thought that the hemiplegia was due to arterial thrombosis or infarction and would imply some other pathologic mechanism. Further investigation along this line is needed.

CASE 1.

A 26 month old colored female developed measles 7 days prior to admission and 1 hour before admission had a sudden onset of ataxia and irritability. She had no other symptoms. Her past history was complicated by a coarctation of the aorta repaired at the age of 5½ months and an intraventricular septal defect repaired at

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the age of 16 months, both with excellent results. The rest of the history was non-contributory.

On admission, her temperature was 101° and pertinent physical findings were limited to the central nervous system, with the exception of a grade II systolic murmur heard over the entire precardium with a loud second pulmonic sound. Examination of the nervous system showed that she had bilateral Babinski's. She was generally flaccid and completely ataxic being unable to sit or stand and a hemiparesis on the left side was noted. The child was extremely irritable and very difficult to manage at the time of admission. Shortly after admission, she was examined by members of the Department of Neurology who concurred with our findings.

A blood count revealed a hemoglobin of 10 grams, 11,200 leucocytes with 23% polynuclear cells, 68% lymphocytes and 5% monocytes. The urinalysis was within normal limits. Spinal fluid on admission had no cells and the protein was 16 mgms. percent; the culture showed no growth. The spinal tap was repeated 2 days after admission and again no cells were present. The total protein was 16 mgms. percent and culture was negative. An electroencephalogram was done and the impression of the neurologist was that this was a tracing compatible with that of a normal child of this age. Skull x-rays were reported as a negative study.

At the time of admission, the impression was that this infant had either encephalitis due to measles virus or a drug intoxication of unknown etiology despite the negative history for the latter. However, it was felt both by the Pediatric and the Neurology Departments that because of the asymmetric neurological findings a drug intoxication or depression was ruled out. The child was observed for approximately 18 hours and then because there was no change in the neurological findings, she was started on a course of 200 mgms. of Cortisone a day for approximately a day and a half. The Cortisone dosage was then tapered slowly over a period of 10 days. There was marked improvement after 36 hours and the child was able to walk without any ataxia. Subjectively, she became very cheerful and cooperative. The hemiplegia had disappeared on examination. She was observed for a period of 48 hours after the steroids had been discontinued and there was no recurrence of any neurological symptoms. She had no other treatment during this time except supportive and prophylactic antibiotics.

The child was seen in the out-patient department one week after discharge and at that time, neurological examination was normal. The family moved away preventing further follow up.

CASE 2.

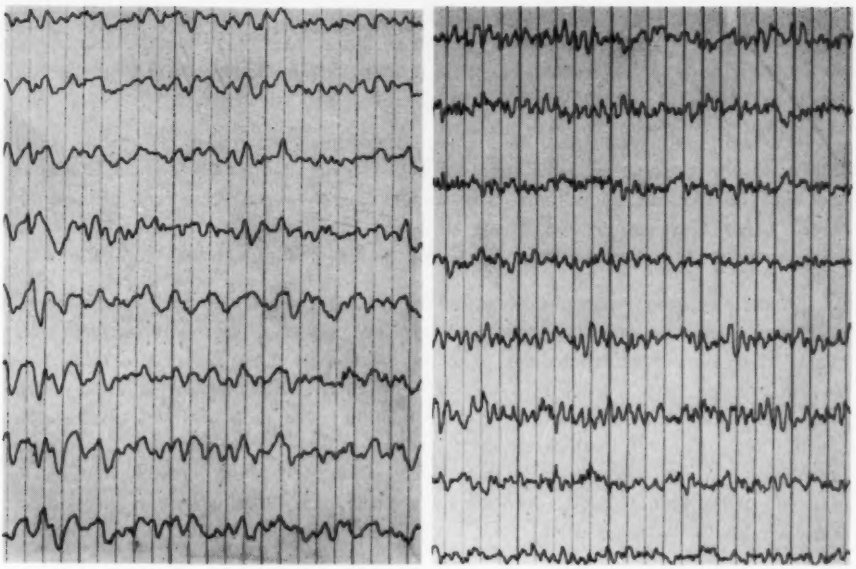
A 6 year old female was admitted to the Henry Ford Hospital on May 1, 1958 with a diagnosis of measles encephalitis. She had developed the rubeola rash 3 days prior to admission and at the time of the eruption, the child was put on prophylactic Penicillin 200,000 units twice a day. She was doing well until the night of admission when suddenly she complained to her mother that there were "flies" over the left side of her body; she became disoriented and had a headache. These complaints persisted for about an hour, the child then vomited and fell into a sleep. She was

seen by one of us 2 hours after the onset of the symptoms at which time she could be aroused. After she was awake from 5 to 10 minutes, it was obvious that the child was not oriented; she was grossly ataxic and quite irritable. She had a 3+ nuchal rigidity. In walking she had a definite weakness of the left leg and arm. Her temperature was 103°. She had a typical rubeola rash with severe conjunctivitis and bronchitis. She was then admitted to the hospital.

On admission the physical findings were as described above. In addition, her neurological examination showed that she had a hyperreflexia of both knee jerks which appeared to be equal. She had a clonus of both ankles. There was a bilateral Babinski. Her eyegrounds were viewed with some difficulty although it was thought that she had a slight amount of papilledema. The pupils were unequal although they both responded to light. Soon after admission, she had a lumbar puncture with an opening pressure of 360 millimeters of water. The child was quiet during the course of the lumbar puncture and the pressure readings were thought to be good. The closing pressure was 180. Five minutes after the lumbar puncture was completed, the child began to have a left sided convulsion which over the next few minutes became generalized; this responded over a 15 to 20 minute period to Phenobarbital and Paraldehyde injections. Spinal fluid revealed 180 cells, all were mononuclear. Her initial white blood count was 21,300 with a left shift. Her hemoglobin was 12.9 grams. The spinal fluid sugar was 70, the chlorides 120, the protein was 38. The child was immediately given 100 milligrams Hydrocortisone IV and then started on Cortisone 300 milligrams a day. This was supplemented by a low salt diet and Potassium Triplex, 2 drams 3 times a day, Maalox and prophylactic Penicillin. Twelve hours after the institution of steroid therapy, there was much improvement. Her sensorium was clearing, she was oriented as to time and place. There was possible left facial weakness and very mild ataxia. The fundi appeared quite normal. Some weakness remained in the left arm and leg.

A day and a half after admission, her neurologic examination was entirely unremarkable. She had a slight speech impediment but the parents stated that she had had this before the onset of the encephalitis. From that point she was completely normal in her physical examination. She was kept on 300 mgms. of Cortisone for 6 days. The dosage was then tapered over a 3 day period, and the patient discharged 10 days after her admission.

Five days after admission and after the onset of the encephalitis, she had an electroencephalogram. Dr. Churchill of the Department of Neurology stated: "The EEG revealed distinct abnormality consisting of difference in activity of the 2 hemispheres, the right being the abnormal one. This is the type of abnormality that is frequently associated with hemiplegias following infection and convulsion in children. Specifically, the right hemisphere showed high amplitude 4/sec. waves that were not seen on the left side. Occasional waves that were quite sharp in outline were observed from the right temporal region." She had a psychometric evaluation for a baseline of future evaluation. The child showed a normal I.Q. of 98 and no manifestations of any behavior disturbance. Her EEG 3½ months later was completely normal, as was her neurological examination.



A B

Figure 1. Electroencephalograms of Case 2. The left curve, A, shows distorted waves before treatment, the right curve B, normal waves after treatment.

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HAMARTOMA*

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One author[1] suggests that the name "hamartoma", coined by Albrecht, should send us to our dictionaries rather than our case records. Dorland's Medical Dictionary will tell you that an hamartoma is a tumor due to overdevelopment of normally placed tissue elements or due to an abnormal relationship of a normally situated tissue element. It is derived from the Greek words meaning "error" and "tumor".

In the past seven years the literature includes 105 articles on hamartoma, chiefly concerned with the pulmonary field: lung, bronchi, and even a bronchial artery hamartoma. Fifty-eight articles deal with this while only 5 are devoted to hamartoma for the gynecologist: 2 ovarian, 1 uterine, 1 vaginal, and 1 unspecified. Ten articles deal with hamartoma in the digestive tract including one in a Meckel's diverticulum. Seven articles deal with its presence in the central nervous system including 3 producing precocious puberty[2] when the hypothalamus was involved. Nine articles consider hamartoma of the kidney, 4 deal with the liver, 3 the heart, 2 the spleen, 2 the skin and one each for hamartomas of the face, neck, parotid gland, mediastinum and hand.

EMBRYOLOGY

The tumors of interest to the gynecologist are mesonephric in origin. Pronephric tubules fuse to form the mesonephric duct. The pronephric duct becomes mesonephric tubules. The paramesonephric duct arises later and its course lies parallel to the mesonephric duct. The oviducts are derived from the lateral portions of the paramesonephric ducts and the medial portions fuse to form uterus and vagina. The mesonephric ducts regress in the female but remnants or vestiges occasionally may be found.

Novak, Woodruff, and Novak[3] have considered tumors of mesonephric origin in ovary, broad ligament, cervix and vagina while Gardner, Greene, and Peckham[4] in 1948 did a comprehensive evaluation of cystic tumors of the broad ligament pointing out that these could be differentiated histologically more accurately than on the basis of location. These authors also were champions of a reform in terminology avoiding the names of individuals in referring to embryologic structures. Subsequent serious authors have agreed in writing but with skepticism in spirit to use these more appropriate terms.

Schiller[5] in 1939 had described mesonephromas of the oavry and Stowe[6] has published a study of these tumors more recently. Because of the relative rarity, much of the literature deals with single case reports. Wolfe[7] in 1940 examined 1,413 cervices, amputated below the level of the internal os at the time of surgery, and found only one example of persistent remnants of mesonephric tissue.

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*Presented before the Wisconsin Society of Obstetrics and Gynecology, Eau Claire, Wisconsin, October 25, 1958.

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Huffman[8] in 1948 gave a succinct series of illustrations of the embryology of the mesonephric structures with his study of mesonephric remnants in the cervix. Wolfe[7] quotes from an article written in 1937 by J. Sakuraoka who found mesonephric rests were "especially frequent in the corpus uteri, generally at the level of the round ligament and in the isthmus at the level of the internal os" in Japanese women. In recent literature in English, cases of hamartoma of the body of the uterus have been limited to a single case report wherein the tumor actually arose from an uterine blood vessel. Hughes[9] points out that the fetal cervix is much longer than the corpus of the uterus making it seem more likely that cervix would be the site of these vestiges rather than the uterus.

MALIGNANT POTENTIAL

To expect all of these tumors to be benign is unjustified. Again isolated cases form the basis for much of the literature, but there are many documented cases of malignant tumors such as clear cell carcinoma of the ovary, solid malignant neoplasm in the broad ligament[10], mesonephric carcinoma of the vaginal fornices and mesonephric adenocarcinoma and clear cell carcinoma arising in the cervix — all believed to be of mesonephric origin. These authors have pointed out that glomeruli are no more necessary to make the diagnosis of a malignant tumor's origin from mesonephric structures than it is necessary to identify Leydig cells to diagnose arrhenoblastoma.

The case that drew local attention to this type of tumor was one in which it was purely an incidental finding. Mrs. C. J., a 43 year old married white gravida 3, para 2, abortus 1, had been seen at Henry Ford Hospital since 1952 with symptoms which were diagnosed as psychosomatic although the patient refused to accept this diagnosis. In 1956 a gynecologist found uterine enlargement and a left adnexal tumor. She developed premenstrual spotting which for a year prior to dilatation and curettage on June 7, 1958, lasted 7 days. The pathologists's report on the endometrial tissue was "proliferative endometrium". When symptoms persisted, hysterectomy was advised two months later.

On August 21, 1958, a vaginal hysterectomy, right salpingo-oophorectomy and cystocele repair were done under Nembutal-nitrous oxide-oxygen anesthesia. The pathologist reported hyperplasia of the endometrium, leiomyoma uteri, chronic cervicitis, hamartoma of the cervix, and right paraovarian cyst.

The histopathologic description of the incidental hamartoma follows: "The slide is from endocervix. No squamous epithelium is present, and a few endocervical glands are dilated and filled with mucus. Deep within the cervix, well beyond the deepest penetration of the endocervical glands, there is a fairly well circumscribed, but non-encapsulated collection of epithelial-like structures in the form of variable sized acini and tubules. The epithelium which lines these spaces shows only a moderate range of variability, from low cuboidal to low columnar. The nuclei are generally round to ovoid with moderate staining nuclear material. The cytoplasm is uniform, clear in general, occasionally eosinophilic and infrequently vacuolated. A few of these spaces are filled with an eosinophilic, amorphous material. A thin reticular sheath surrounds these structures which lie within a stroma composed

primarily of fibrous tissue of varying density with occasional strands of smooth muscle noted."

No other cases were included in our index, but a second case has since been reported. A similar description was made, with these exceptions: location was in the portia as squamous epithelium was identified in the same section. A larger number of the spaces contained eosinophilic material. Several areas distant from the adenoma-like collection contained single or grouped minute tubule-like structures with histological similarity to both of the previous lesions described. It is believed that these structures are minute mesonephric remnants.

DISCUSSION

The history and physical examination are of no value in making this diagnosis before surgery. It is entirely an histopathologic diagnosis made by the thorough pathologist who seeks or at least is aware of these mesonephric remnants. Their reported incidence varies from 1 in 1,413 to 40%. A recent study by Sneeden[11] in which the remnants were actually sought produced an incidence of 7.7% to dispute the estimate of 1% made by Huffman and supported by Novak and others.[3] In order to recognize the cancers of varied microscopic appearance that develop from this lesion, the lesion itself must be recognized.

CONCLUSION

It is necessary for us as practicing gynecologists to know something of hamartomas or tumors of mesonephric origin so we can adequately explain them to our patients when an alert pathologist makes the diagnosis: "Hamartoma".

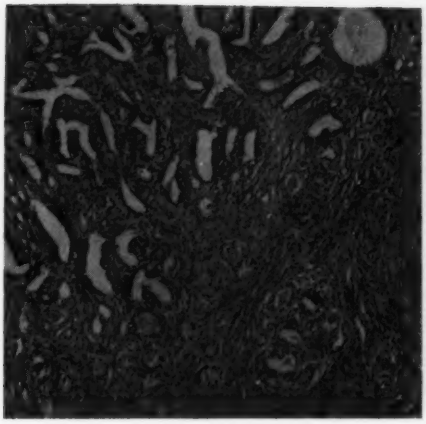

Figure 1

Case No. 1 — Specimen of the Hamartoma X 70.

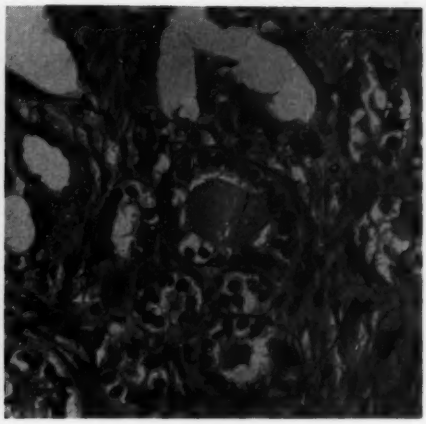

Figure 2

Case No. 1 — Specimen of the Hamartoma X 300.

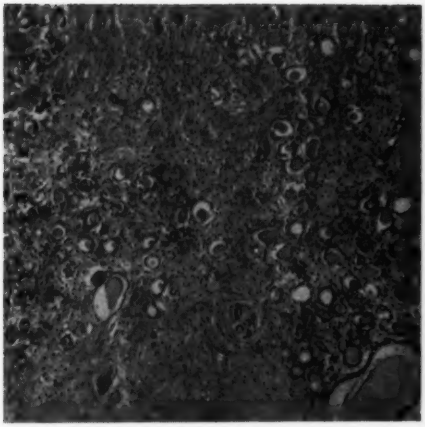

Figure 3

Case No. 2 — Specimen of the Hamartoma X 70.

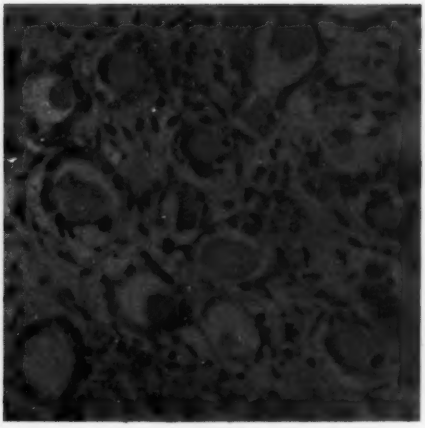

Figure 4

Case No. 2 — Specimen of the Hamartoma X 300.

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EXPERIMENTAL STUDIES WITH LOW OXYGEN FLOW RATES IN THE BUBBLE OXYGENATOR

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Accumulated clinical and experimental experience with extra-corporeal circulation has led to the solution of many of the problems which initially confronted workers in this field. However, in many centers where cardio-pulmonary by-pass is utilized for open heart surgery, a certain number of "cerebral deaths" have occurred early in the postoperative period. This syndrome frequently presents a fairly constant pattern of an immediate lucid interval followed by agitation, depression, coma and death occurring 12 to 72 hours postoperatively. This complication has been observed to follow the use of several types of oxygenators. Many causal factors have been suggested and investigated. Although oxygen toxicity and gas embolism have not been demonstrated as etiologic agents responsible for this syndrome, it is entirely reasonable to suspect these causes and attempt to eliminate or minimize them.[1] The investigations herein reported were designed to establish the minimum oxygen flow rates in the DeWall-Lillehei bubble oxygenator which would produce satisfactory oxygenation of the blood and at the same time prevent excessive amounts of physically dissolved oxygen in the plasma. Unphysiologic arterial oxygen partial pressures would thereby be avoided in the perfused blood, which if sufficiently high, could produce gas embolism. Additional advantages would consist of decreased trauma to the blood in the oxygenator and simplified defoaming in the debubbling chamber.

Clark and co-workers[2] have reported the polarographic monitoring of blood oxygen tension as a means to insure adequate oxygenation while avoiding over saturation of the perfused blood. Since it has been determined that the plasma oxygen partial pressure does not exceed 200 mm. of Hg. (normal 100 mm. of Hg.) until 98.8% saturation is reached, excessive oxygen tensions would be avoided by saturation levels below this point.[3,4] Provided that oxygenator efficiency could be approximately determined, a suitably low oxygen flow rate could be used in the oxygenator which would produce saturation levels sufficient to supply body oxygen needs and result in a more physiologic perfusion. A constant oxygen tension monitoring device would then be unnecessary.

Accordingly, a series of experiments were designed to establish if possible, a relationship between optimal oxygenation, oxygen flow rates related to body weight, perfusion rate and size of oxygenator components.[5] Total cardio-pulmonary by-pass was carried out on several series of dogs weighing between 9 and 15 kilograms. The

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DeWall-Lillehei unit and Sigmamotor pump were utilized in all experiments. Anesthesia was induced with intraperitoneal Nembutal and exposure obtained through a bilateral trans-sternal, fourth interspace thoracotomy. Arterial inflow was through the left subclavian artery. Cannulae in the inferior and superior vena cavae were brought out through the right atrium and drained into a reservoir by gravity. Aortic, central venous pressures, rectal temperature, and blood pH were monitored. Oxygen saturations were determined by photoelectric cell and content by VanSlyke method. Samples for these studies were drawn simultaneously from arterial and venous lines at periodic intervals during the by-pass. During the course of each perfusion, the endotrachael tube was occluded. Oxygen metering valves having a maximum flow rate of eight liters/min. were utilized for accuracy in measuring the small flow rates.

RESULTS

In the first series of experiments, (Table 1) 10 dogs were perfused at blood flow rates of 30, 50, and 70 cc/kg./min., and oxygen flow rates of 2, 3, 5, and 7 liters/min., for each liter of blood flow. In reviewing the results of this group, normal

Bl. Flow		O_2 Flow		Temp.		B.P.		Art.		Ven.		
cc/Kg/min.	Total	Ratio	Total	Rectal	Water bath	Art. mm.Hg.	Ven. Cm.H_2O	O_2 Cont.	Sat.	O_2 Cont.	Sat.	A-V diff.
30	420	2/1	1.0	94	110	50	-5	16.1	86	8.1	43	8.0
30	420	5/1	2.0	93	110	48	-1	17.9	95	7.8	41	10.1
30	420	7/1	3.0	94	112	57	-1	17.8	95	7.1	38	10.7
50	700	2/1	1.5	94	107	90	∱6	15.8	84	8.6	46	7.2
50	700	5/1	3.5	94	112	85	-1	17.7	94	9.6	51	8.1
50	700	7/1	5.0	93	110	85	0	18.4	98	10.8	58	7.8
70	980	2/1	2.0	94	112	105	∱3	17.3	92	11.7	62	5.6
70	980	5/1	5.0	93	113	98	-1	18.6	99	13.4	71	5.2
70	980	7/1	7.0	93	110	100	∱1	18.6	99	15.6	83	3.0

Table I

Table 1

Representative data obtained from 10 perfusion experiments which were carried out at various blood and oxygen flow rates.

arterial and venous oxygen saturations (and therefore normal A-V differences) occurred at a perfusion rate of 70 cc./kg./min., with a ratio of 2.2 liters of oxygen for each liter of blood flow (ratio of 2.2:1). Normal arterial oxygen saturation levels were produced by a perfusion rate of 50 cc./kg./min., and oxygen flow rates of 3:1. Mean arterial pressure at the lower perfusion rate was 80 mm. Hg., while a rate of 70 cc./kg./min., resulted in pressures of at least 100 mm. Hg. The mixing column was 2.5 x 50 cm., and the defoamer 5 x 60 cm.

A second group of experiments was carried out in an identical manner but the perfusion period was extended to 2 hours in order to detect development of an oxygen debt. Satisfactory arterial oxygen saturation levels of 93 to 96% were again noted with perfusion rates of 50 and 70 cc./kg./min., with 3:1 and 2.2:1 oxygen flow rates respectively. With this longer perfusion, however, a progressively increasing A-V oxygen saturation difference was noted with the lower blood perfusion rate. This indicates that the amount of oxygen transported by the blood in this circumstance was insufficient to supply body requirements. A progressive oxygen debt developed. This observation was not made when a blood flow rate of 70 cc./kg./min., was used. When oxygen flow rates above 2.2:1 were used with a blood perfusion rate of 70 cc./kg./min., the arterial oxygen saturation reached the 98-100% range and the venous saturation exceeded 80%. The A-V oxygen saturation difference decreased progressively with time, indicating the utilization of dissolved plasma oxygen by the perfused animal.

These investigations demonstrate that there is a low optimal oxygen flow rate for the oxygenator which can be related to the body weight of the perfused subject. This oxygen flow rate is significantly lower than that commonly in use with the bubble oxygenator. This value was repeatedly determined to be between 150 and 180 cc. of oxygen/kg. of body weight of the perfused subject. Bubble size, and oxygenator length (which determines the length of time blood is exposed to oxygen at the liquid-gas interface) are factors which determine efficiency of the apparatus and must be standardized. As a check on these observations, cardiopulmonary by-pass was performed using a continuous oxygen flow rate of 150 cc./kg. body weight while the blood flow rate was increased to levels which maintained normal aortic pressures. As might be expected, perfusion rates in the latter range (70-80 cc./kg./min.) were productive of the most physiologic oxygenation (Table 2) in the arterial and venous blood. *A 30% increase in oxygen transportation occurs when blood flow is increased from 50 to 70 cc./kg./min.*

The pH of the blood was usually low during cannulization and steadily increased to normal values during the perfusion when the higher blood flows were used. Carbon dioxide tension levels remained in the physiologic range during use of the 2.2:1 oxygen flow rates. Plasma hemoglobin levels were below 100 mgm. and fibrinogen determinations within normal limits under these circumstances. Debubbling was simplified due to a lack of foaming with the low oxygen flow rates.

SUMMARY AND CONCLUSIONS

Short of an ideal perfusion technique which has as yet to appear, a reasonable goal would be a perfusion which resulted in physiologic values for the readily

Bl. Flow			O_2 Flow	Temp.		B. P.		Art.		Ven.		
cc./Kg/min.	Total	Ratio	Total	Rectal	Water bath	Art. mm.Hg.	Ven. Cm.H_2O	O_2 Cont.	Sat.	O_2 Cont.	Sat.	A-V diff.
50	650	2/1	1.3	94	108	80	-5	17.9	87	11.2	57	6.7
50	650	3/1	2.0	94	108	75	-6	19.4	86	11.7	59	7.7
50	650	4/1	2.5	93	110	95	-4.5	20.8	97	10.0	50	10.0
50	650	5/1	3.0	93.5	110	100	∱3	23.1	99	12.9	61	10.2
50	650	6/1	3.9	93.5	108	120	∱7	22.2	95	15.5	75	6.7
70	910	2/1	2.0	93	110	90	-4	19.7	88	13.7	66	6.0
70	910	3/1	2.5	93	112	100	-3	20.7	96	15.0	70	5.7
70	910	3.5/1	3.0	93	110	115	-3.5	21.7	94	15.1	72	6.6

Table II

Table 2

Perfusion data obtained by varying the blood flow rate while total oxygen flow remains constant.

measurable blood constituents. Normal and occasionally above normal blood flow rates will be required to meet the preoperative cardiac output of the perfused subject.

In an attempt to approach physiologic perfusion, abnormally high arterial oxygen tensions which might lead to gas embolism should be avoided. Satisfactory oxygen saturation levels unaccompanied by excessive oxygen partial pressures were obtained with surprisingly low oxygen flow rates (2.2:1) in the bubble oxygenator. These results were attained only when using blood flow rates of at least 70 cc./kg./min. Under these conditions, the arterial-venous oxygen saturation difference remained constant during long perfusion periods. The latter probably constitutes a critical test of oxygen transportation and utilization. The principles derived from these studies have been applied to clinical perfusions with very satisfactory results.

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GALLSTONE ILEUS

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DOUGLAS M. EVANS, M.D.*

Report of three cases and a brief review of the literature.

It is the purpose of this report to stress some of the clinical manifestations of the mechanical obstruction of the gastrointestinal tract caused by gallstones; this clinical and pathological entity of which the infreqeuncy has been overemphasized, occurs in 1 to 2% of all cases of non-strangulating intestinal obstruction. Although the mortality from intestinal obstruction has been reduced to 5 to 7% recent reports still show a mortality from gallstone ileus of 40 to 50%[1]. This cholelithic obstruction of the alimentary canal is considered to be a potentially dangerous complication of the so-called "silent gallstone". In this paper, we review three cases seen in the last three years at the Henry Ford Hospital, in which the diagnosis was proved at operation or by passage of a gallstone through the rectum, thereby relieving the obstruction.

HISTORICAL DATA

Bartholin in 1654[2] published a case of perforation of a gallstone from the gallbladder into the jejunum. Curvoisier[3], however, was the first one and justly deserves the credit for having directed surgical endeavor into the proper channels. He published 131 cases which constituted a masterful production. Subsequently, numerous reports have appeared. In 1914, Wagner[4] collected 334 cases from the literature. Moore[5] estimated the number as about 400 in 1925, later, in 1942, Foss and Summers[1] collected an additional 150 cases; and, according to Deckoff[2] in 1955, the number of recorded cases of gallstone ileus at that time was 555.

It is very obvious that for gallstone ileus to occur, a gallstone must be first formed. Most frequently, this takes place within the gallbladder, although it may form in the extrahepatic biliary system which is very unusual. It is of interest that gallstone ileus has been reported when the gallbladder was not present.[6]

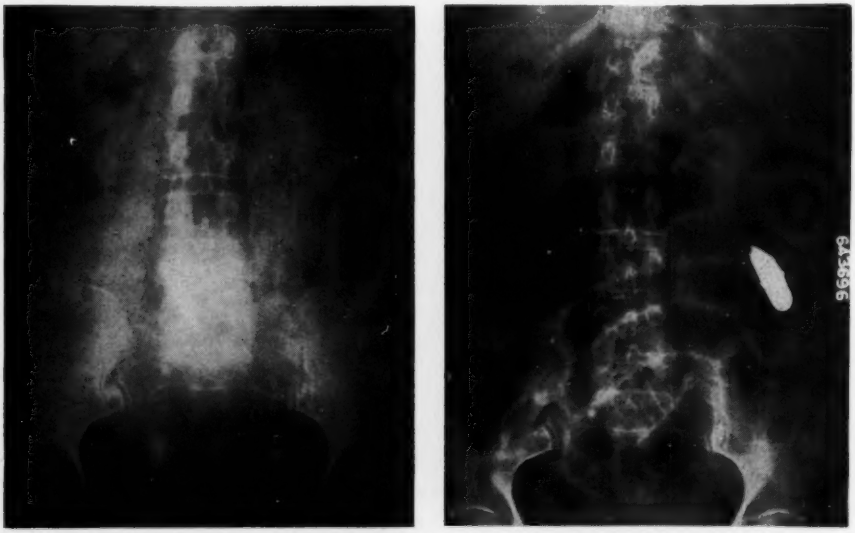
Following the formation, the stone may obstruct the alimentary tract in one of two ways: 1. Entering the peritoneal cavity. Inflammation about it may then cause kinking and extra luminal obstruction of the intestine, however. 2. More often the blockage is caused by entrance of the stone into the lumen of the bowel producing an obturation type of obstruction. The latter may be caused by three different methods: 1. A gallstone free in the peritoneal cavity may ulcerate into the intestine, sometimes long after perforation of the gallbladder had occurred. 2. The stone may enter the duodenum via the common duct and ampulla of vater. This has been a very controversial point. Murphy[7] reported a case in which the ductus choledochus was dilated enough to permit the passage of a stone measuring 4.5 cms. 3. Almost always, the presence of gallstone ileus is associated with a cholecystoenteric fistula. Such a

*Department of Surgery.

fistulous tract connects the gallbladder with the duodenum in the vast majority of the cases but the colon, stomach and jejunum are connected with the gallbladder less frequently. In addition, rarities connecting the gallbladder and the pleural and pericardial cavities, the pregnant uterus and the renal pelvis have been reported. It is not the purpose of this paper to describe the pathogenesis of these fistulae which has been described in the past by Borman and Rigler in 1937.[8]

CLINICAL CASES

First Case. J. B. No. 643696, a 60 year old white female was seen in the emergency room at the Henry Ford Hospital in acute distress with the chief complaint of right upper quadrant and epigastric pain of sudden onset 4 days prior to admission to the hospital. This pain was stabbing in nature and localized to the previous mentioned areas up to the moment of admission, after which it became more generalized in the right side of the abdomen. Anorexia, nausea and vomiting accompanied the pain with the onset of the symptoms. The patient became progressively distended and obstipated. No flatus or bowel movement had been noted for the past 4 days. The past history disclosed evidences of previous "gallbladder attacks" and myxedema, produced several years ago by the administration of radioactive iodine. On physical examination, the appearance of the patient was that of an elderly woman, obese, with the classical myxedematous appearance. Vital signs were T. 99.4° F. Pulse 98, Respiration 18 and blood pressure 118/84. ENT examination was negative. Lungs were clear to percussion and auscultation. Heart tones normal with a regular rhythm; no presence of murmur or gallop. The abdomen was distended with no scars visible. The patient was tender in the right upper quadrant and right flank on palpation. The epigastrium was also moderately tender. The abdomen was tympanitic and bowel



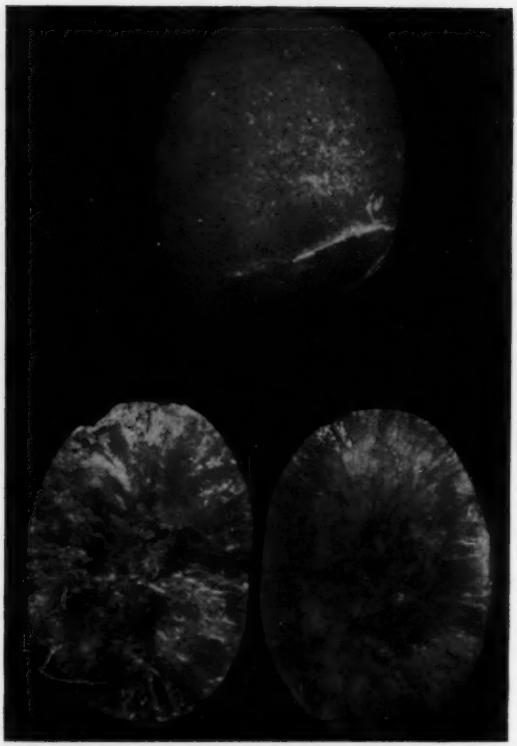
Case No. 643696 J. B. Flat Plates of the abdomen shows on: A. taken on day of admission marked distension of the jejunum, and air in biliary tree. B. 2nd day after admission distension increase, special polyethilene tube with the mercury bag at level of the ligament of Treitz.

sounds were present but markedly hypoactive. Rectal and pelvic examinations were non-contributory. Laboratory work on admission was as follows: Diastase 110%, W.B.C. 6450, Hgb. 14.2 gms., urine negative, chest x-ray normal. The flat plates of the abdomen showed evidence of ileus and air in the region of the gallbladder and the biliary tree but no stone was identified within the abdominal cavity. Conservative therapy consisted of intestinal intubation and suction, parenteral fluid and electrolyte replacement. On the first and second hospital days the patient's condition remained the same. Seen by endocrinologist and Cytomel is given. The repeat x-ray films of the abdomen showed persistence of the air in the area of the biliary tree and no gas in the large bowel. On the fourth hospital day, a barium enema was obtained and showed diverticulosis with no evidence of diverticulitis or malignancy. The patient,

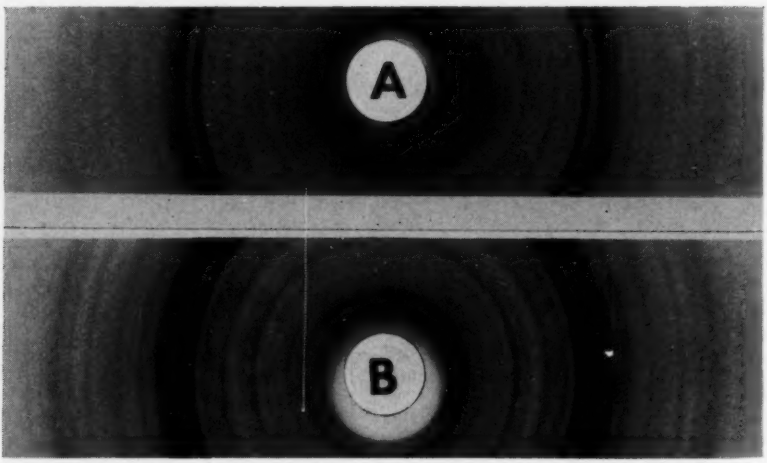
Case No. 643696 J. B. Flat Plates of the abdomen shows on: the 3rd day after admission long polyethilene tube used for suction with mercury bag near the terminal ileum, small bowel dilatation with improvement — residual of barium enema shows diverticulosis.

in the afternoon, on expelling the barium with a saline enema, passed a large ovoid stone measuring 1¼ by ½ inches. This stone proved to be more than 90% cholesterol when examined by the x-ray difraction method at the Edsel B. Ford Institute, Department of Physics.

After the passage of the stone, the patient underwent a frank period of recovery. Cholecystograms eleven days after the initial admission showed non-function of the gallbladder and the presence of a round density which represented another gallstone. Upper gastrointestinal series disclosed the presence of a cholecystoduodenal fistula. Patient was discharged on a bland diet and Cytomel for out-patient department follow-up.



Stone from Case No. 643696, J. B. Passed spontaneously per rectum on 6th day after admission. This proved to be 95% pure cholesterol.



Patterns produced by the x-ray difraction of the stone. A. Normal pattern of cholesterol and — B — the pattern of the stone from Case No. 643696 — J. B.

Case No. 643696 J. B.: A. Barium enema shows transverse colon, the gallbladder area seen with a calcified stone and evidences of air. B. shows cholecystograms demonstrates non function of gallbladder and the calcified and laminated stone. C and D. upper gastrointestinal serial films, with demonstration of the cholecysto-duodenal fistulous tract.

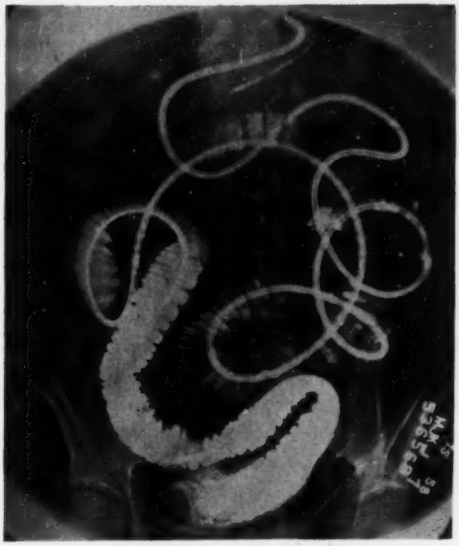
Second Case. D. N. No. 819746 age 75. White male admitted via the emergency room with the chief complaint of abdominal pain, crampy in nature and diffuse, nausea and vomiting with marked obstipation for the last six days. There was a history of previous biliary colic. Laboratory data on admission was non-remarkable. Abdominal distention was present, diffuse tenderness with presence of hyperperistalsis. Rectal examination disclosed benign prostatic hypertrophy. The x-ray films of the abdomen showed distended loops of jejunum with the presence of aberrant or migratory calculi within the abdominal cavity. The calculi shifted from the left

Case No. 819746 D. N. The presence of jejunal distension is very striking. There is a stone on the abdominal cavity on the first film (A) The stone lies on the L.L.Q. — on the second film (B) taken the next day, the stone is in the R.L.Q. — also a stone is seen on the area of the gallbladder.

lower quadrant in the first set of films to the right lower quadrant on the second ones. Intestinal intubation was accomplished and suction instituted in addition to the electrolyte and fluid restoration. Antibiotics were given. 48 hours after admission the patient was taken to the operating room and a gallstone was removed from the jejuno-ileal junction measuring 3 to 3.5 inches in diameter. It was broken in two pieces and it was observed that the stone served as a nucleus for the deposit of stool all the way around it. The patient had a stormy recovery, including phlebothrombosis occuring on the 7th postoperative day. An acute hemorrhagic prostatitis followed, which subsided with medication and finally the patient was discharged in good condition.

Third Case. K. H. 63 year old white female, first seen at the Henry Ford Hospital in April of 1948, at the age of 53, with complaints of upper abdominal bloating and flatulence. Jaundice, melena and vomiting were denied at the time. The objective findings were mild hypertension, cystocele and rectocele with obesity. A clinical diagnosis of biliary tract disease was made at the time. While cholecystograms were being obtained, the patient did have a marked allergic reaction to the dye and was admitted for treatment of this. The films at the time showed a poor filling of the gallbladder with a large radio-opaque shadow suggesting cholelithiasis. Biliary drainage showed three plus cholesterol crystals. Surgery was advised but the patient refused. In view of this, she was placed on a low fat diet and appropriate medications. She was followed in the out-patient department for ten years. She presented herself in the emergency room on June 30, 1958, with a two day history of intermittent cramping abdominal pain and vomiting. The pain began in the epigastrium then shifted to the

periumbilical area. No bowel movement had been noted for the last several days and no passage of gases per rectum for the last 24 hours. Initial examination showed the abdomen to be soft with bowel sounds hypoactive. No hernia or any abdominal scars were noted. The blood pressure was 120/76, Hgb. 14.2, W.B.C. 12,000, F.B.S. 85, N.P.N. .38, Diastase 119%. Urine analysis negative. An initial diagnosis of small bowel obstruction due to diverticulitis was made. Conservative therapy was initiated, including Miller-Abbott tube suction and intubation of the small intestine and parenteral fluids. Clinical Course: When the patient did not show any evidence of resolution of the process at the fifth day of conservative measures, a barium enema was obtained on July 5, 1958. No diverticuli or tumor were present. An attempt to restore oral intake was made with the subsequent return of crampy abdominal pain. Intestinal suction and parenteral fluids were reinstituted with symptomatic relief but no signs of restitution of bowel function. During this interval, the patient displayed no elevation of temperature or pulse and no abdominal findings of an intraperitoneal, localizing process. In view of the presence of persistent obstruction of the small bowel due to a non-inflammatory process, upper gastrointestinal x-ray films were obtained. A calcific density was demonstrated, obstructing the flow of barium. A question of



Case No. 536568 K. H. Administration of barium through the Miller-Abbot tube outlines the jejunum and ileum; suddenly stops at the place of the obstruction, where a round density is outlined.

barium in the biliary tree was raised. A diagnosis of gallstone ileus was made and the next day the patient was taken to the operating room. A huge stone was removed from the ileum; resection of this portion had to be done for trauma produced by the stone. The postoperative convalescence was uneventful. Patient was discharged on the 11th postoperative day and followed in the out-patient department where she continued to do well.

On September 14, 1958, the patient was readmitted to the hospital and on September 16, 1958, returned to the operating room where a cholecystectomy, common

bile duct exploration and closure of the cholecystoduodenal fistula was done. The recovery was uneventful and follow-up to date showed the patient to be in good condition.

Case No. 536568 K. H. Ileum shows the place and bulging area where the stone was lodged. Notice the local trauma and venous ingurgitation. A resection of this portion of the bowel was undertaken.

COMMENTS

In our first case, J. B., the diagnosis of gallstone ileus was suspected on admission to the emergency room and became strongly supported when, on some of the progress films, there was seen the presence of air in the area of the biliary tree. In retrospective analysis, the most likely possibility is that the stone was caught in the terminal ileum with the long axis of the stone in a transverse position in relation to the lumen of the bowel and, as has been stated by Foss and Summers,[1] 70% of these stones get caught in this area. The fact that the patient did obtain relief after the barium enema was due to the fact that reflux of barium through the ileocecal valve dislodged the stone from the original place. From the work of Murphy[7] and Wagner,[4] it is known that the stones pass spontaneously through the rectum in the vast majority of the cases.

In view of the myxedematous condition of the patient and the lack of any clinical manifestations in relation to the gallbladder and biliary tract duct system, it was elected to follow the case conservatively.

The second case also was admitted through the emergency room. The diagnosis was elicited and confirmed on admission. Once the patient was prepared and suitable, the surgical procedure was carried out very successfully. Once more the stone was in the jejuno-ileal junction. This patient refused final surgical treatment to the cholecysto-intestinal fistulous tract and we have not seen him since that time.

In the third case, the patient was known to have gallbladder disease in the past. However, the first clinical impression was that of diverticulitis and, only when the

barium enema was taken, was this entity ruled out and it was decided to subject the patient to a small bowel x-ray examination which showed the place of the blockage and the stone causing it. A classical sequence of events followed. First, the removal of the stone and, a month later, the final treatment for the cholecystoduodenal fistula, namely, cholecystectomy, common bile duct exploration and closure of the duodenum.

SYMPTOMATOLOGY

In general, these cases are very confusing and they are often misdiagnosed. This is the main reason for delay in therapy, with the consequent increase in mortality for this condition. A past history suggestive of cholelithiasis is found in just about 60% of the cases.[9] We did have it in the three cases reported here. This is not always obtained. The probable reason for this low frequency is the fact that gallstone ileus is often the complication of the so-called "silent stone". Symptoms of acute cholecystitis preceding the onset of the ileus is also a very uncommon situation according to Foss and Summers.[1] Only in 25% of the cases is it seen. In our cases, there were none of these acute manifestations. Jaundice also was not present in any of the cases here reported and in Deckoff's[2] series, only one of his patients had icterus.

The initial episode may simulate biliary colic, acute cholecystitis or diverticulitis and may even resemble acute pancreatitis. In some cases there is a diagnosis of perforation of the gallbladder. The preliminary symptoms of small bowel obstruction may occur later, after the stone has passed into the bowel. Clute[10] reported a case in which the symptoms did not appear until five years later. As the stone starts to travel in the lumen of the bowel, it causes partial, intermittent obstruction with cramps, nausea and vomiting. The nature of the delayed symptoms with spontaneous and sometimes with long periods of relief, influences the victim to delay medical treatment. With the final lodging of the stone, the signs and symptoms become acute, distention occurs, vomiting increases with obstipation being the main feature. In some cases, the patient may still pass some flatus and excrete feces from below the place of obstruction and then the clinical diagnosis of the condition is very difficult. The appearance of stones in the vomitus or in the feces is pathognomonic[11].

DIAGNOSIS

Preoperative diagnosis is infrequently made, mainly because most of the patients are aged, acutely and chronically ill and a good history is difficult to elicit. However, when there is a past history of gallbladder disease in an elderly female, obese and suffering with small bowel obstruction in whom abdominal surgery has not been done and no herina is present, the diagnosis of gallstone ileus should be strongly considered.[12] The abdominal findings usually are helpful to the diagnosis. Abdominal distention is present, although it may only be revealed by roentgenogram. The temperature and leucocytic count are normal or within normal limits. The average temperature of our cases was 99 and the leucocytic counts ranged from 5000 to 8000. The most valuable aid in the diagnosis is the roentgenologic examination of the abdomen. Flat, upright and lateral films of the abdomen should be taken and, in addition, spot films of the liver region in several positions.

The presence of distended loops of bowel either jejunum or ileum, may suggest the place of obstruction. In other cases, the stone may be seen in addition to the air in the biliary tree. In some other cases, like the third case, the ingestion of barium sulphate may outline the stone. A barium enema may outline the stone which is obstructing the colon. Finally, a gallstone ileus may be suspected if a previous film of the gallbladder shows stone and this is not present any more on subsequent examinations. The differential diagnosis includes all causes of intestinal obstruction such as hernia, adhesions, intussusception, tumor, mesenteric thrombosis, etc. Acute pancreatitis, acute diverticulitis, renal and biliary colics should be ruled out.

TREATMENT

Surgical intervention is the treatment of choice soon after the patient's condition has been restored to optimal, including water and electrolyte replacement. The use of the long intestinal tubes for decompression of the small bowel is of great and primary help in treatment preoperatively and postoperatively.

SUMMARY

Intestinal obstruction due to a gallstone, an infrequent condition, should be kept in mind in order to achieve a diagnosis in all the cases of intestinal obstruction. Awareness of this entity will produce a clinical diagnosis in many unsuspected cases. The diagnosis of gallstone ileus is suggested when the following findings are present: A. Intestinal obstruction in an obese, elderly patient, more often a female. B. Past history of gallbladder disease. C. Abdomen free of surgical scarring. D. Roentgenological evidences of air in the biliary tree and the presence of a calculus anywhere in the abdominal cavity. The treatment in the acute phase of the obstruction should be directed to produce decompression of distended bowel, restoration of fluids and electrolytes and blood volumes. Once the patient has been properly prepared, the treatment of choice is the removal of the stone unless it is passed in natural ways.

The final treatment of the condition where a cholecystoenteric fistula exists should be evaluated in each individual case since many of these patients will not tolerate an extensive procedure. The age of the patient and presence of associated diseases plays a very important role in the final decision. In the cases where surgery is indicated, the treatment of choice is cholecystectomy, common bile duct exploration and closure of the fistulous opening in the enteric tract. It should be possible to reduce the mortality of this condition from the high rate of 40%-50% to a point similar to the actual one accepted for simple intestinal obstructions; 2 to 7%. The ideal treatment is the prophylaxis by removal of gallbladders which contain stones.

A series of three cases is presented with no mortality.

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NEW STAFF MEMBER — 1958

William L. Morgan, Jr., M.D. joined our staff in Cardiology on August 1, 1958. He was born in Honolulu, Hawaii and is 29 years of age. He received his B.A. degree from Yale University in 1948. He received his M.D. degree from Harvard University in 1952. He served one year of internship and three years of medical residency at the Massachusetts General Hospital, taking one year of training in Cardiology during his residency. He then served two years with the National Heart Institute at Bethesda, Maryland in special research on Cardiovascular Hemodynamics. He resides at 16821 East Jefferson, Grosse Pointe with his wife Joan and five month old daughter Nancy.

ERRATUM

In the article by Hurley and Shelley in our September, 1958 issue, page 284, the heading of the second column from the right should read Silica. The word Colloidal should be deleted.

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Published quarterly by the Professional Staff of the
Henry Ford Hospital and The Edsel B. Ford Institute for Medical Research

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HENRY FORD HOSPITAL MEDICAL BULLETIN

Report from The Edsel B. Ford Institute for Medical Research

Vol. 6 December, 1958 No. 4
Part II

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Henry Ford Hospital
1958

X-RAY DIFFRACTION POWDER DATA AND INDEX FOR THE STEROIDS

JONATHAN PARSONS*, WILLIAM T. BEHER** AND GIZELLA D. BAKER**

X-ray diffraction as an analytical tool is well established in the fields of mineralogy, metallurgy and inorganic chemistry. The technique has not been used widely in the study of biological materials, particularly organic materials, even though many organic compounds have a crystalline nature.

The study of atherosclerosis has been pursued in the biochemistry department under a grant from the Michigan Heart Association. In 1954 the authors felt that x-ray diffraction powder techniques could be applied to identifying the steroids found in aorta plaques and also in chemical extracts from organs of experimental animals. Although the steroids as a group produce good x-ray diffraction patterns, a set of standard patterns would first have to be provided in order to use this comparison technique. The only x-ray diffraction studies previously done on a number of steroids were those of Bernal and Crowfoot[1,2] and Bernal, Crowfoot and Fankuchen[3]; these were studies by single crystal methods, which supplied no data which could be used for powder identification.

During the last four years x-ray diffraction powder data has been procured by the authors for 137 steroids obtained from pharmaceutical supply houses and the Chemistry Department, Wayne State University. The data for these steroids were published in four papers appearing in Analytical Chemistry[4,5,6,7].

The need for specific crystallization procedures became apparent as several definitely different patterns were observed for different samples of the same steroid. Different polymorphic forms of a specific compound may exist; the form present in any single case is generally due to the crystallization conditions. The steroids used for the first papers[4,5] were not recrystallized. Inquiry was made from the supply sources about the crystallization procedures but in many cases the latter information was not available. It was decided to recrystallize all steroids under controlled and recorded conditions. Some changes in the pattern data originally published have resulted from these recrystallizations.

Lest those not previously acquainted with x-ray diffraction powder analysis gain the impression that there are no stumbling blocks in applying the technique to the analysis of biological extracts, the authors wish to point out that this is unfortunately not the case. There are certain limitations about which a laboratory considering the adoption of these methods should be made aware. These limitations and difficulties in applying this technique have been fully considered by Frevel[8] and will be only briefly mentioned here.

Any unknown steroid of biological origin is likely to be composed of more than one steroid component. Many members of this family are isomorphic, differing from

*Edsel B. Ford Institute, Department of Physics.
**Edsel B. Ford Institute, Department of Biochemistry.

one another only in minor side chain structure or in the physical properties of individual atoms leading to intensity differences in lines which can be diffracted from corresponding planes of atoms. It is quite common to find the relative intensities (I/I.) of the lines of an unknown steroid considerably altered from the reported relative intensities of the pure steroid. This fact might lead to difficulty or even failure in the use of the Hanawalt Table (Table II).

Another effect which must be watched for when isomorphism is present is the effect of the formation of solid solutions. When the type of structure and the dimensions of related compounds are the same or very similar, corresponding atoms may interchange positions or be substituted for each other. To the extent that such substitutions take place, there will often be slight changes in relative atomic positions which will appear as slight shifts in the diffracted line positions. It is possible for a solid solution to have a diffraction pattern differing considerably from that of either separate component. Specific examples of this effect have been considered by Davis[9].

In enumerating the above complications in the biological application of x-ray diffraction powder analysis, it is not intended to under-estimate the potential value of this technique in biology and medicine. It is hoped that the availability of this and other similar sets of standard powder pattern data will serve to initiate new research studies in this and allied medical fields.

In preparing this material in its present form, it has been the purpose of the authors (1) to give a brief description of x-ray diffraction, (2) to provide an index to facilitate identifications of steroids from powder pattern data, (3) to record under one cover all the steroid pattern data obtained to date in this laboratory. Permission has been granted by Analytical Chemistry to use some data as originally published by them.

DESCRIPTION OF X-RAY DIFFRACTION POWDER METHOD

When a narrowly collimated beam of monochromatic x-rays is incident upon a crystal, regular deviation (diffraction) of the incident beam occurs. The intensity and direction of the diffracted beam will depend upon the wave-length of the x-rays used, the geometric arrangement of the atoms in the crystal, and also the number of electrons in the individual atoms themselves.

A schematic drawing of a conventional powder camera is shown in Figure 1. The powdered specimen, inserted in a tube, is placed on the axis of the cylindrical body of the camera (X). The filter, used to make the x-ray source as nearly monochromatic as possible, is shown at F; the tube containing the apertures which collimate the x-ray beam is shown at C. The collimated x-ray beam, on passing through a group of small randomly oriented crystals, will be diffracted into surface cones having varying semi-vertical angles 2θ. Each ray on the surface of any particular cone is diffracted by a particular set of planes in a single crystal. All crystal planes at any particular angle θ to the original beam, but oriented at various positions around the beam direction, will form the cone of semi-vertical angle 2θ. The sample tubes are rotated upon their axis in order to insure better random orientation.

Even a simple crystal, like sodium chloride, will contain many planes when diagonal as well as surface planes are included. Different sets of crystal planes with their discrete interplanar spacings, d-values, will give rise to separate cones. These coaxial cones, with their vertices at the specimen position (X), have their angular sizes determined by the distances between various atomic planes in the crystal.

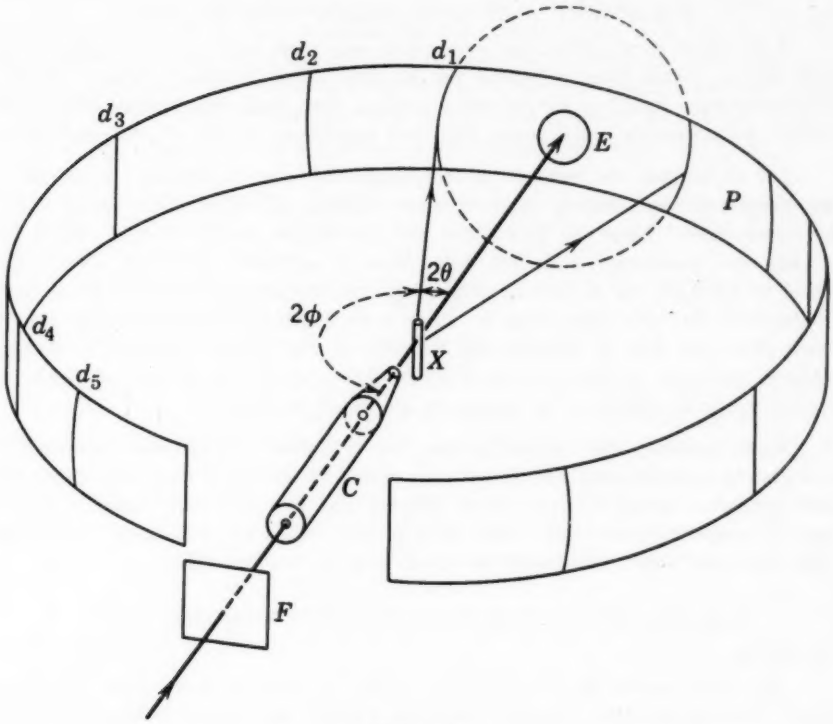

Figure 1

Geometrical features of the x-ray diffraction powder camera.

The pattern obtained when a photographic film is placed on the cylindrical inside surface of the powder camera will be a series of dark rings, such as d_1, d_2, and d_3 (Figure 1). These rings will be concentric with the original beam direction. When measurements are made upon the flattened out film, from the various rings to the exit port position, values are obtained which are functions of the interplanar spacings (d-values) which give rise to the separate rings. The mathematical relationship known as Bragg's Law is given by the equation $n\lambda = 2d \sin \theta$ where n is the order of reflection, λ is the wave-length of the x-rays, d is the interplanar spacing between successive atomic planes in the crystal, and θ the angle between the atomic plane and both the incident and reflected beams. The geometry of the 114.59 mm powder

camera is such that 2θ can be easily determined for each ring by measuring along the film from ring to the non-diffracted beam position on the film (exit port position). No computation using the above formula is necessary, since several sets of tables[10] and charts[11] are available to convert directly from film measurement to interplanar spacings (d-values). For a complete description of the x-ray diffraction powder method of analysis authoritative discussions are listed with the references[12,13,14].

EQUIPMENT AND OPERATIONAL PROCEDURES

A Norelco* x-ray diffraction source unit was used, fitted with a copper target tube with a nickel filter to isolate the K*a* line. Two Norelco* powder cameras (Debye-Scherrer type), of 114.59 mm diameters, were used. The normal settings for voltage, milliamperage, and exposure time were respectively 35 KV, 20 ma and 5 hours.

For all steroids, the methods of recrystallization, were as follows: A sample of the compound was dissolved in a minimum volume of solvent while being heated on a steam bath. Water was then added until the solution became cloudy. While this solution was maintained at boiling temperature, a sufficient amount of solvent was added to redissolve the precipitate. The compound was allowed to crystallize at room temperature. In those cases where the above method failed to yield satisfactory results, ethyl ether was used to enhance the solubility of the steroid. Solvents used were ethanol, methanol, acetone and ethyl ether. The majority of the steroids were recrystallized from ethanol. The exceptions are noted in Table I.

When possible, after recrystallization, the crystalline steroids were powdered in a mortar to approximately 200 mesh†. The powdered specimens were then introduced into capillaries having 0.5 mm inside diameter and 0.01 mm wall thickness. These special capillaries‡ are made from glass having low x-ray absorption coefficient. They have the added convenience of funnel ends to facilitate filling.

DESCRIPTION OF TABLES AND PATTERN PHOTOGRAPHS

TABLE I:

This table consists of a classified list of the 137 steroids included in the present paper. The melting point of each compound reported has been determined§, checked with the literature, and listed in a separate column. The numerical index number assigned to each steroid, serves to identify the compound in the following tables and for the pattern photographs.

TABLE II.

In 1938, Hanawalt, Rinn and Frevel[15], proposed a system of powder pattern identification which is used, in a modified form, by the American Society for Testing Materials for publishing the "X-ray Powder Data File[16]. The original Hanawalt plan has been followed in preparing this table.

---

*North American Philips Co., Inc., 750 South Fulton Avenue, Mt. Vernon, New York

†Size of screen having 200 wires to the inch.

‡Available from Caines Sales Co., 3020 N. Cicero Avenue, Chicago 41, Illinois.

§Fisher-Johns Melting Point Apparatus.

The Hanawalt system of powder pattern identification is based upon the measurement of d-values of the three most intense lines of each pattern. The three strongest lines of each pattern are indexed three times according to the spacing (d), namely in the order 1-2-3, 2-1-3, and 3-1-2 where 1,2,3 represnts the relative intensities of the three strongest lines. The first d-value of each of the above orders determines the Hanawalt group in which the three lines are placed. The values of the second lines in descending order are used to determine the sequence of listing within each Hanawalt group; should the d-values of two second position lines be the same, then the descending order of the third position lines are used. On each line in the table the three d-values are printed in sequence followed by the three corresponding values of I/I_1, in the same order, and then followed by the reference number of the steroid. The key to these reference numbers is to be found in Table I. The Hanawalt group number and range of d-values is found at the beginning of each group.

To illustrate the method of listing, androstane -3, 17-dione (Ref. No. 45) will be used as an example. The three strongest lines for this steroid (see Table III) are (1) d=5.42 A*, $I/I=_1 1.00$; (2) d=4.94 A, $I/I_1=0.67$; and (3) d=6.42 A, $I/I_1=$ 0.49. The first listing, 5.42, 4.94, 6.42 (order 1-2-3), is found in Hanawalt group No. 70; the second listing, 4.94, 5.42, 6.42 (2-1-3), is found in group No. 68; the third listing, 6.42, 5.42, 4.94 (3-1-2), is found in Group No. 73.

In using this numerical index the three strongest lines of the "unknown" steroid are determined and a visual estimate of their relative intensities is made. Choose the Hanawalt Group that includes the d-value of the strongest line. Now proceed to look for the second strongest line in the second column of the group table. If successful, the third d-value in the table will be found within the region of the third d-value of the "unknown" pattern. Allowances must be made for errors in d-value measurements. For this reason, Hanawalt Groups above and below the first one consulted should be examined if the strong line d-value is near the first or last of the Hanawalt Group range. Errors in the determination of relative intensities are the reasons for the alternate listing sequences in the table. Should the first search in the group with order 1-2-3 be fruitless, proceed to search the table with alternate relative intensity orders, i.e., 2-1-3 and 3-1-2. When particular steroid possibilities have been found using this table, the agreement of the other pattern lines should be checked by consulting the complete d-value listing of Table III.

TABLE III:

This table consists of the interplanar spacings, d-values, for each steroid, measured in angstrom units* (A). Also included in this table are the relative intensities of each diffracted line. The darkest (most intense) line of each pattern is given an arbitrary intensity rating of 1.00, with other lines ranging down the relative scale. The listed intensities (I/I_1) can be related only to other lines from the same pattern and have no absolute intensity significance. The relative intensity measurements were made using a technique of graded intensity scale comparison, developed by Hanawalt, Rinn and Frevel[15] and modified by Klug and Alexander[13].

*One angstrom unit equals one ten millionth of a millimeter.

PATTERN PHOTOGRAPHS:

The authors feel that the inclusion of photographs of patterns is desirable in presenting diffraction data even though some lack of uniformity and intensity distortion may result from the reproductions of the original patterns. A quick preliminary identification of a steroid can often be made by comparison of the pattern photographs, without taking pattern measurements. The key to the pattern photographs is to be found in Table I.

ACKNOWLEDGMENT

The cooperation and encouragement of Dr. John H. L. Watson, Head of Physics Department, and Dr. O. H. Gaebler, Head of Biochemistry Department of the Edsel B. Ford Institute for Medical Research, are greatly appreciated.

Many thanks to the following individuals for their time and efforts in contributing to this paper: Dr. Carl Djerassi, Chemistry Department, Wayne State University, who kindly donated some of the steroids used in this study; Mr. John Kroll, Photographic Department, Henry Ford Hospital, for the photographic reproductions of the x-ray diffraction patterns; Dorothy Hebert, Kathleen Matheson, and Marion Tessin, for technical assistance in film measurement and data tabulation.

The authors wish to thank *Analytical Chemistry* for permission to use some of the tables and photographs previously published.

Figure 1 has been reprinted from *X-ray Diffraction Procedures*[13] with permission from the authors and John Wiley and Sons, Inc.

The authors are grateful to Professor I. Fankuchen and Dr. Dan McLachlan, Jr., who read the manuscript and offered helpful suggestions for its final revision.

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16. *"X-ray Powder Data File"*, published by the American Society for Testing Materials, 1916 Race St., Philadelphia 3, Pennsylvania.

Table I. Index to Steroid X-Ray Diffraction Powder Data

Index No.	Name	Melting Point (Uncorr.), °C
	C_{18} Steroids	
1	$\Delta^{1,3,5}$-Estratriene-3, 17α-diol[a]	176-179
2	$\Delta^{1,3,5}$-Estratriene-3, 16, 17-triol	280-282
3	$\Delta^{1,3,5,6,8}$-Estrapentaene-3-ol-17-one	257-261
4	$\Delta^{1,3,5}$-Estratriene-3-ol-17-one	259-261
5	1-Methyl-$\Delta^{1,3,5}$-estratriene-3-ol-17-one	251-256
6	$\Delta^{1,3,5}$-Estratriene-3-ol-17-one-methyl ether	167-169.
	Monochloro	
7	3β-Chloro-Δ^{5}-cholestene[a]	92-94
	Monohydric Alcohols	
8	Androstane-17-ol	164.5-166.5
9	Cholestane-3α-ol	182-184.5
10	Cholestane-3β-ol	141-142
11	Cholestane-3β-ol-3-acetate	108-109
12	Δ^{5}-Cholestene-3β-ol[a]	146-148
13	Δ^{5}-Cholestene-3β-ol-3-acetate[a]	115-116
14	$\Delta^{5,7,22}$-Ergostatriene-3-ol	ca 160
15	Neosterol (75.5% Ergosterol, 24.5% $\Delta^{7,22}$-Ergostadiene-3β-ol)[a]	164-168
16	$\Delta^{5,22}$-Stigmastadiene-3β-ol	167-169
17	$\Delta^{5,22}$-Stigmastadiene-3β-ol-3-acetate	140-141.5
18	Δ^{5}-Stigmastene-3β-ol	138-140
19	Δ^{5}-Stigmastene-3β-ol-3-acetate	125-127
	Dihydric Alcohols	
20	Allopregnane-3β, 20α-diol-3-acetate	168-169
21	Allopregnane-3β, 20β-diol-diacetate	141-145
22	$\Delta^{5,7}$-Androstadiene-3β, 17β-diol[b]	Dec. 225-228
23	$\Delta^{5,7}$-Androstadiene-3β, 17β-diol-dibenzoate	218-220
24	Δ^{5}-Androstene-3β, 17β-diol	180-182
25	Δ^{5}-Androstene-3β, 17-diol-diacetate	160-162
26	Δ^{5}-Androstene-3β, 17β-diol-3-acetate-17-benzoate	176-177
27	Δ^{5}-Androstene-3β, 17β-diol-17-benzoate[a]	218-220
28	Δ^{5}-Androstene-3, 17-diol-17-cyanide	Dec. 233-234
29	17-Ethyl-Δ^{5}-androstene-3, 17-diol	193-195
30	17-Ethynyl-Δ^{5}-androstene-3, 17-diol	245-246
31	17α-Methyl-Δ^{5}-androstene-3β, 17β-diol	204-205
32	Δ^{5}-Androstene-3, 17-diol-17-propionate	148-152
33	Δ^{4}-Cholestene-3β, 6β-diol-diacetate	133.5-135
34	Pregnane-3α, 20α-diol	237-239
35	Pregnane-3α, 20β-diol	232.5-235
	Trihydric Alcohol	
36	24, 24-Dimethyl-Δ^{5}-cholene-3β, 20, 24-triol	194-196
	Monoketones	
37	Δ^{16}-Allopregnene-20-one	155.5-157.5
38	Androstane-17-one	115-120
39	Cholestane-3-one[a]	128-129
40	Cholestane-3-one enol acetate	92-97
41	Δ^{1}-Cholestene-3-one	103-106

Index No.	Name	Melting Point (Uncorr.), °C
42	Δ^{4}-Cholestene-3-one	80-82
43	Coprostane-3-one[a]	60-63
	Diketones	
44	Allopregnane-3, 20-dione	202-205
45	Androstane-3, 17-dione	131-133
46	Androstane-3, 17-dione-3-enol acetate	145-147
47	Δ^{1}-Androstene-3, 17-dione	137-139
48	Δ^{4}-Androstene-3, 17-dione	172-174
49	Pregnane-3, 20-dione	117-120
50	Δ^{4}-Pregnene-3, 20-dione[a]	120-122
	Monohydroxymonoketones	
51	Allopregnane-3β-ol-20-one	192-195
52	Allopregnane-3β-ol-20-one-3-acetate	144-146
53	Androstane-3α-ol-17-one[a]	184-185
54	Androstane-3β-ol-17-one	172-173.5
55	Androstane-17β-ol-3-one	175-178
56	Androstane-17-ol-3-one-17-acetate	157-159
57	Androstane-17β-ol-3-one-17-benzoate	194-196
58	Androstane-17β-ol-3-one-17-hexahydrobenzoate	161-163
59	Δ^{4}-Androstene-17α-ol-3-one	152-159
60	Δ^{4}-Androstene-17-ol-3-one-17-acetate-3-enol acetate	151-154
61	Δ^{4}-Androstene-17-ol-3-one-17-benzoate[b]	212-214
62	17β-Ethynyl-Δ^{4}-androstene-17α-ol-3-one	282-284
63	17β-Methyl-Δ^{4}-androstene-17α-ol-3-one	161-164
64	Δ^{4}-Androstene-17β-ol-3-one-17-propionate	117.5-120
65	Δ^{4}-Androstene-17-ol-3-one-17-propionate-3-enol propionate[a]	123-126
66	Δ^{5}-Androstene-3β-ol-17-one	151-153
67	Δ^{5}-Androstene-3β-ol-17-one-3-acetate	168-170
68	Δ^{5}-Androstene-3β-ol-17-one-3-acetate-17-cyanide[a]	205-209
69	Δ^{5}-Androstene-3β-ol-17-one-3-methyl ether	137.5-140.5
70	Etiocholane-17β-ol-3-one-17-acetate	146-148
71	Pregnane-3β-ol-20-one-3-acetate	112-115
72	Δ^{4}-Pregnene-20β-ol-3-one	166-168
73	Δ^{5}-Pregnene-3β-ol-20-one	191-193
74	Δ^{5}-Pregnene-3β-ol-20-one-3-acetate	149-151
75	16, 17-Oxido-Δ^{5}-pregnene-3β-ol-20-one-3-acetate	155-158
76	Δ^{5}-Pregnene-3β-ol-20-one-3-methyl ether	125-127
77	$\Delta^{5,16}$-Pregnadiene-3β-ol-20-one	173-175
78	$\Delta^{5,16}$-Pregnadiene-3β-ol-20-one-3-acetate	173-175
	Dihydroxymonoketones	
79	Allopregnane-3, 21-diol-20-one-21-acetate	201-203.5
80	Pregnane-3α, 17α-diol-20-one	215-218.5
81	Δ^{5}-Pregnene-3β, 17-diol-20-one	272-276
82	Δ^{5}-Pregnene-3β, 21-diol-20-one-21-acetate[a]	183-185
83	Δ^{5}-Pregnene-3β, 21-diol-20-one-3, 21-diacetate	163-165
84	16, 17-Oxido-Δ^{5}-pregnene-3β, 21-diol-20-one-21-acetate	185-189
	Trihydroxymonoketones	
85	Allopregnane-3β, 17α, 21-triol-20-one-21-acetate[d]	231-235
86	Δ^{4}-Pregnene-17α, 20b, 21-triol-3-one-20b, 21-diacetate	188-190.5
87	Δ^{5}-Pregnene-3β, 17, 21-triol-20-one-3, 21-diacetate	267-270

Index No.	Name	Melting Point (Uncorr.), °C
	Tetrahydroxymonoketone	
88	Pregnane-3*a*, 17*a*, 11β, 21-tetrol-20-one[a]	216-218
	Monohydroxydiketones	
89	Allopregnane-21-ol-3, 20-dione-21-acetate	194-196
90	Δ^4-Pregnene-17-ol-3, 20-dione	215-217
91	Δ^4-Pregnene-21-ol-3, 20-dione	138-141
92	Δ^4-Pregnene-21-ol-3, 20-dione-21-acetate	155-157
93	16, 17-Oxido-Δ^4-pregnene-21-ol-3, 20-dione-21-acetate	166-168
	Dihydroxydiketones	
94	Pregnane-17*a*, 21-diol-3, 20-dione-21-acetate[a]	195.5-197.5
95	Δ^4-Pregnene-17*a*, 21-diol-3, 20-dione	207-210
96	Δ^4-Pregnene-17*a*, 21-diol-3, 20-dione-21-acetate	235-237.5
	Dihydroxytriketones	
97	Δ^4-Pregnene-17*a*, 21-diol-3, 11, 20-trione[a]	222-225
98	Δ^4-Pregnene-17*a*, 21-diol-3, 11, 20-trione-21-acetate[a]	236-240
	Ethers	
99	i-Cholesterol methyl ether	79-83
100	Bis-cholesteryl ether[c]	196-199
	Carboalkoxy Acids	
101	3β-Acetoxybisnorallocholanic acid	191-194
102	3β-Hydroxy-Δ^5-bisnorcholenic acid	291-293
103	3β-Acetoxy-Δ^5-bisnorcholenic acid	234-236
104	3*a*-Hydroxy-cholanic acid	185-187
105	3, 6-Dihydroxy-cholanic acid	----
106	3, 12-Dihydroxy-cholanic acid	171-173
107	3, 12-Diformoxy-cholanic acid	193-195
108	3, 7, 12-Trihydroxy-cholanic acid	196-198
109	3, 12-Diketo-cholanic acid	185-186.5
110	3, 7, 12-Triketo-cholanic acid	234-236
111	3-Formoxy-12-keto-cholanic acid	200-203
112	3β-Hydroxy-Δ^5-cholenic acid	240-242
113	Diacetoxynordeoxycholic acid	205-207
	Carboalkoxy Esters	
114	Methyl diacetylbisnordeoxycholate	164-167
115	Methyl 3, 12-dihydroxycholanate[d]	98-102.5
116	Methyl 3, 12-diacetoxycholanate[d]	114-116
117	Methyl 3-acetoxy-12-ketocholanate	145-148
118	Methyl 3-benzoxy-12-ketocholanate	123.5-126
119	Methyl 3-keto-12β-acetoxycholanate	118-119
120	Methyl diacetylnordeoxycholanate	152-156
	Sapogenins and Sapogenin Derivatives	
121	Δ^5-Androstene-3β, 17β-diol-17-benzoate digitonide[a]	----
122	Δ^5-Cholestene-3β-ol- digitonide[a]	----
123	Δ^5-Stigmastene-3β-ol- digitonide[a]	----
124	Diosgenin acetate[a]	195-197
125	Hecogenin	261-262
126	Δ^4-Hecogenone	Dec. 233-235
127	Isosarsasapogenin acetate	151-152

Index No.	Name	Melting Point (Uncorr.), °C
128	Kryptogenin acetate	148-150
129	Sarsasapogenin	198-201
130	Smilogenone	181-186
	Digitalis Glycosides	
131	Digitoxin[a]	245-249
132	Digoxin[a]	260-265
133	Gitoxin[a]	Dec. 282
	Steroid Derivatives	
134	Methyl bisdehydrodoisynolic acid	231-235
135	Deoxybilianic acid	295-298
	Pseudo Steroids	
136	Meso-3, 4-bis-(p-hydroxyphenyl)-n-hexane	182-184
137	3, 4-Bis-(p-hydroxyphenyl)-3-hexene	169-170

FOOTNOTES TO TABLE I

[a]Acetone
[b]Ethanol and ethyl ether
[c]Ethyl ether
[d]Methanol
[e]Not recrystallized

Table II. Hanawalt Numerical Index

d(A)			I/I_1			Reference No.*
		85. 17.9 to 16.0				
17.0	5.17	5.59	30	100	50	10
		84. 15.9 to 14.0				
14.0	6.15	5.29	40	100	53	122
14.7	5.96	4.95	27	100	50	101
15.6	5.95	5.45	55	100	73	14
15.0	5.54	4.42	20	100	20	125
14.1	5.31	5.93	27	100	55	102
14.5	4.62	5.55	53	100	73	32
		83. 13.9 to 12.0				
12.0	6.28	8.98	28	100	56	106
13.5	5.14	6.46	75	100	50	136
		82. 11.9 to 11.0				
11.2	5.81	4.66	56	100	73	91
11.2	5.79	4.65	50	100	50	86
11.8	5.57	4.24	27	100	40	37
11.0	5.47	5.88	41	100	27	100
11.6	5.32	5.83	55	100	73	35
		81. 10.9 to 10.0				
10.5	5.49	4.66	53	100	53	7
10.4	5.42	4.77	50	100	75	13
10.4	5.37	4.46	27	100	37	99
10.3	4.72	5.40	15	100	41	40
		80. 9.99 to 9.50				
9.66	5.84	4.85	27	100	73	79
9.66	5.54	4.95	27	100	67	39
9.99	5.17	4.54	49	100	100	67
		79. 9.49 to 9.00				
9.35	7.63	4.97	73	100	86	120
9.21	5.83	4.91	53	100	53	105
9.07	5.41	5.11	53	100	53	57
		78. 8.99 to 8.50				
8.76	6.64	5.29	75	100	50	119
8.98	6.28	12.0	56	100	28	106
8.71	4.55	6.17	75	100	90	114
		77. 8.49 to 8.00				
8.19	5.55	4.71	73	100	73	118
8.04	5.45	4.89	49	100	83	115
8.33	5.24	6.96	50	100	63	50
8.04	4.82	5.01	74	100	74	75

d(A)			I/I$_1$			Reference No.*
			76. 7.99 to 7.50			
7.63	4.97	9.35	100	86	73	120
7.56	4.78	6.24	100	86	73	113
			75. 7.49 to 7.00			
7.31	6.20	5.65	53	100	100	117
7.02	6.15	5.02	75	100	92	127
7.38	6.07	5.14	56	100	65	116
7.44	5.93	4.71	27	100	73	16
7.37	5.89	4.93	36	100	67	72
7.20	5.89	4.89	55	100	86	107
7.06	5.85	5.14	37	100	75	4
7.47	5.83	4.72	55	100	73	60
7.23	5.32	5.59	49	100	49	96
7.28	5.20	4.68	27	100	37	68
7.03	4.87	6.19	49	100	36	17
			74. 6.99 to 6.50			
6.64	8.76	5.29	100	75	50	119
6.76	6.26	4.42	53	100	53	69
6.81	5.95	4.58	37	100	37	43
6.54	5.83	5.02	73	100	73	8
6.84	5.80	5.28	15	100	20	80
6.78	5.74	6.49	75	100	75	108
6.71	5.64	4.78	88	100	88	1
6.74	5.49	4.98	35	100	73	41
6.89	5.47	3.74	37	100	37	42
6.96	5.24	8.33	63	100	50	50
6.57	5.15	4.31	100	73	41	71
6.49	5.10	5.57	75	100	75	135
6.51	5.08	4.73	100	67	67	111
6.71	4.90	5.54	56	100	75	5
6.66	4.85	5.99	67	100	82	89
6.17	4.55	8.71	90	100	75	114
6.73	4.34	6.05	73	100	30	2
			73. 6.49 to 6.00			
6.28	8.98	12.0	100	56	28	106
6.24	7.56	4.78	73	100	86	113
6.26	6.76	4.42	100	53	53	69
6.17	6.33	5.77	100	73	73	78
6.33	6.17	5.77	73	100	73	78
6.19	5.93	5.39	75	100	75	129
6.01	5.83	5.06	100	100	75	81
6.01	5.80	5.54	100	49	49	70
6.28	5.79	5.56	100	73	73	49
6.37	5.75	5.38	53	100	53	31
6.49	5.74	6.78	75	100	75	108

d(A)			I/I_1			Reference No.*
6.01	5.74	5.16	100	100	56	54
6.07	5.74	5.10	100	100	59	76
6.44	5.74	4.30	40	100	53	30
6.11	5.72	5.21	73	100	40	124
6.15	5.72	5.04	100	27	27	123
6.13	5.68	4.62	100	73	73	55
6.08	5.66	5.23	100	100	100	61
6.20	5.65	7.31	100	100	53	117
6.09	5.64	5.31	75	100	88	63
6.07	5.63	5.15	100	73	53	93
6.09	5.59	5.91	40	100	30	44
6.11	5.59	4.93	100	64	64	95
6.17	5.57	4.98	100	40	40	21
6.12	5.56	5.25	40	100	53	20
6.07	5.55	4.68	100	73	73	52
6.01	5.54	5.20	100	100	88	59
6.21	5.52	4.17	100	100	83	128
6.03	5.51	4.17	73	100	100	23
6.09	5.49	4.71	100	67	67	64
6.19	5.47	5.25	100	50	37	36
6.42	5.42	4.94	49	100	67	45
6.24	5.40	4.94	53	100	53	46
6.07	5.38	5.75	100	56	38	29
6.39	5.35	4.75	100	100	100	47
6.05	5.32	4.30	100	100	50	27
6.22	5.31	4.71	100	75	56	97
6.15	5.31	4.34	73	100	87	15
6.15	5.29	14.0	100	53	40	122
6.09	5.29	5.11	67	100	100	132
6.42	5.26	4.24	75	100	56	134
6.26	5.21	4.32	40	100	20	33
6.44	5.17	3.72	20	100	53	3
6.01	5.16	4.24	100	75	56	48
6.46	5.14	13.5	50	100	75	136
6.07	5.14	7.38	100	65	56	116
6.01	5.13	5.56	100	100	75	103
6.09	5.11	5.79	100	100	88	11
6.49	5.10	5.57	75	100	75	135
6.07	5.10	4.86	100	53	40	19
6.49	5.05	5.79	100	100	67	133
6.09	5.05	3.87	100	100	75	121
6.15	5.02	7.02	100	92	75	127
6.49	5.02	5.49	100	100	87	58
6.24	5.02	4.67	100	100	94	62
6.19	4.94	5.93	50	100	37	98
6.19	4.87	7.03	36	100	49	17
6.24	4.82	5.39	100	75	50	84
6.11	4.78	5.74	20	100	53	9
6.09	4.75	5.64	73	100	53	83
6.22	4.72	5.87	100	75	50	109
6.17	4.55	8.71	90	100	75	114
6.05	4.34	6.73	30	100	73	2
6.13	4.27	5.49	27	100	27	56

d(A)			I/I$_1$			Reference No.*
		72. 5.99 to 5.75				
5.79	11.2	4.65	100	50	50	86
5.83	9.21	4.91	100	53	53	105
5.95	6.81	4.58	100	37	37	43
5.83	6.54	5.02	100	73	73	8
5.79	6.49	5.05	67	100	100	133
5.75	6.37	5.38	100	53	53	31
5.79	6.28	5.56	73	100	73	49
5.87	6.22	4.72	50	100	75	109
5.93	6.19	5.39	100	75	75	129
5.77	6.17	6.33	73	100	73	78
5.79	6.09	5.11	88	100	100	11
5.75	6.07	5.38	38	100	56	29
5.80	6.01	5.54	49	100	49	70
5.83	6.01	5.06	100	100	75	81
5.97	5.72	4.85	100	100	100	82
5.95	5.72	4.71	100	100	73	66
5.85	5.70	4.42	100	75	37	94
5.99	5.65	5.10	100	40	40	85
5.91	5.59	6.09	30	100	40	44
5.87	5.58	5.39	100	100	64	24
5.85	5.54	4.82	40	100	100	38
5.87	5.54	4.72	100	100	100	104
5.88	5.47	11.0	27	100	41	100
5.97	5.47	4.69	100	73	73	88
5.97	5.47	4.95	100	100	43	73
5.95	5.45	15.6	100	73	55	14
5.83	5.32	11.6	73	100	55	35
5.93	5.31	14.1	55	100	27	102
5.87	5.31	4.64	100	100	40	130
5.80	5.28	6.84	100	20	15	80
5.84	5.17	4.93	100	37	37	126
5.85	5.14	7.06	100	75	37	4
5.87	4.98	4.53	100	73	55	87
5.96	4.95	14.7	100	50	27	101
5.93	4.94	6.19	37	100	50	98
5.89	4.93	7.37	100	67	36	72
5.91	4.90	5.45	100	30	20	90
5.89	4.89	7.20	100	86	55	107
5.84	4.85	9.66	100	73	27	79
5.99	4.85	6.66	82	100	67	89
5.85	4.85	4.45	55	100	73	137
5.91	4.81	4.61	100	58	36	18
5.91	4.80	5.11	100	47	39	53
5.93	4.76	5.08	100	100	37	25
5.76	4.76	3.95	100	73	55	65
5.83	4.72	7.47	100	73	55	60
5.93	4.71	7.44	100	73	27	16
5.81	4.66	11.2	100	73	56	91
5.87	4.42	5.14	75	100	50	34
5.95	4.32	5.63	100	73	53	131
5.79	3.44	5.07	100	73	40	6

d(A)			I/I$_1$			Reference No.*
		71. 5.74 to 5.50				
5.54	15.0	4.42	100	20	20	125
5.55	8.19	4.71	100	73	73	118
5.74	6.78	6.49	100	75	75	108
5.64	6.71	4.78	100	88	88	1
5.56	6.28	5.79	73	100	73	49
5.52	6.21	4.17	100	100	83	128
5.65	6.20	7.31	100	100	53	117
5.57	6.17	4.98	40	100	40	21
5.72	6.15	5.04	27	100	27	123
5.68	6.13	4.62	73	100	73	55
5.72	6.11	5.21	100	73	40	124
5.59	6.11	4.93	64	100	64	95
5.59	6.09	5.91	100	40	30	44
5.66	6.08	5.23	100	100	100	61
5.63	6.07	5.15	73	100	53	93
5.74	6.07	5.10	100	100	59	76
5.55	6.07	4.68	73	100	73	52
5.54	6.01	5.80	49	100	49	70
5.54	6.01	5.20	100	100	88	59
5.74	6.01	5.16	100	100	56	54
5.56	6.01	5.13	75	100	100	103
5.65	5.99	5.10	40	100	40	85
5.72	5.97	4.85	100	100	100	82
5.72	5.95	4.71	100	100	73	66
5.63	5.95	4.32	53	100	73	131
5.58	5.87	5.39	100	100	64	24
5.54	5.87	4.72	100	100	100	104
5.70	5.85	4.42	75	100	37	94
5.74	5.44	4.22	100	100	90	77
5.59	5.33	4.20	67	100	36	92
5.59	5.32	7.23	49	100	49	96
5.64	5.31	6.09	100	88	75	63
5.66	5.26	4.96	100	37	37	110
5.74	5.26	4.91	73	100	41	12
5.56	5.25	6.12	100	53	40	20
5.59	5.17	17.0	50	100	30	10
5.57	5.10	6.49	75	100	75	135
5.55	5.01	4.65	100	100	73	28
5.54	4.95	9.66	100	67	27	39
5.54	4.90	6.71	75	100	56	5
5.70	4.89	4.21	100	53	53	51
5.68	4.87	2.89	87	100	38	112
5.54	4.82	5.85	100	100	40	38
5.74	4.78	6.11	53	100	20	9
5.70	4.78	4.45	73	100	53	26
5.64	4.75	6.09	53	100	73	83
5.55	4.62	14.5	73	100	53	32
5.74	4.30	6.44	100	53	40	30
5.57	4.24	11.8	100	40	27	37
5.51	4.17	6.03	100	100	73	23
5.74	3.63	5.50	75	100	75	74
5.50	3.63	5.74	75	100	75	74

d(A)			I/I$_1$			Reference No.*
		70. 5.49 to 5.25				
5.47	11.0	5.88	100	41	27	100
5.49	10.5	4.66	100	53	53	7
5.41	9.07	5.11	100	53	53	57
5.32	7.23	5.59	100	49	49	96
5.47	6.89	3.74	100	37	37	42
5.29	6.64	8.76	50	100	75	119
5.49	6.49	5.02	87	100	100	58
5.26	6.42	4.24	100	75	56	134
5.35	6.39	4.75	100	100	100	47
5.40	6.24	4.94	100	53	53	46
5.39	6.24	4.82	50	100	75	84
5.31	6.22	4.71	75	100	56	97
5.25	6.19	5.47	37	100	50	36
5.47	6.19	5.25	50	100	37	36
5.29	6.15	14.0	53	100	40	122
5.49	6.09	4.71	67	100	67	64
5.38	6.07	5.75	56	100	38	29
5.32	6.05	4.30	100	100	50	27
5.47	5.97	4.69	73	100	73	88
5.47	5.97	4.95	100	100	43	73
5.45	5.95	15.6	73	100	55	14
5.31	5.93	14.1	100	55	27	102
5.39	5.93	6.19	75	100	75	129
5.45	5.91	4.90	20	100	30	90
5.39	5.87	5.58	64	100	100	24
5.31	5.87	4.64	100	100	40	130
5.32	5.83	11.6	100	73	55	35
5.28	5.80	6.84	20	100	15	80
5.38	5.75	6.37	53	100	53	31
5.26	5.74	4.91	100	73	41	12
5.44	5.74	4.22	100	100	90	77
5.26	5.66	4.96	37	100	37	110
5.31	5.64	6.09	88	100	75	63
5.33	5.59	4.20	100	67	36	92
5.25	5.56	6.12	53	100	40	20
5.47	5.21	4.75	100	100	100	22
5.29	5.11	6.09	100	100	67	132
5.49	4.98	6.74	100	73	35	41
5.42	4.94	6.42	100	67	49	45
5.45	4.89	8.04	100	83	49	115
5.42	4.77	10.4	100	75	50	13
5.40	4.72	10.3	41	100	15	40
5.37	4.46	10.4	100	37	27	99
5.31	4.34	6.15	100	87	73	15
5.49	4.27	6.13	27	100	27	56
		69. 5.24 to 5.00				
5.14	13.5	6.46	100	75	50	136
5.24	6.96	8.33	100	63	50	50
5.15	6.57	4.31	73	100	41	71
5.08	6.51	4.73	67	100	67	111

d(A)			I/I_1			Reference No.*
5.05	6.49	5.79	100	100	67	133
5.10	6.49	5.57	100	75	75	135
5.02	6.49	5.49	100	100	87	58
5.21	6.26	4.32	100	40	20	33
5.02	6.24	4.67	100	100	94	62
5.02	6.15	7.02	92	100	75	127
5.04	6.15	5.72	27	100	27	123
5.11	6.09	5.79	100	100	88	11
5.05	6.09	3.87	100	100	75	121
5.23	6.08	5.66	100	100	100	61
5.14	6.07	7.38	65	100	56	116
5.10	6.07	5.74	59	100	100	76
5.15	6.07	5.63	53	100	73	93
5.10	6.07	4.86	53	100	40	19
5.06	6.01	5.83	75	100	100	81
5.16	6.01	5.74	56	100	100	54
5.13	6.01	5.56	100	100	75	103
5.20	6.01	5.54	88	100	100	59
5.16	6.01	4.24	75	100	56	48
5.10	5.99	5.65	40	100	40	85
5.08	5.93	4.76	37	100	100	25
5.11	5.91	4.80	39	100	47	53
5.14	5.85	7.06	75	100	37	4
5.17	5.84	4.93	37	100	37	126
5.02	5.83	6.54	73	100	73	8
5.07	5.79	3.44	40	100	73	6
5.21	5.72	6.11	40	100	73	124
5.17	5.59	17.0	100	50	30	10
5.01	5.55	4.65	100	100	73	28
5.21	5.47	4.75	100	100	100	22
5.11	5.41	9.07	53	100	53	57
5.11	5.29	6.09	100	100	67	132
5.01	4.82	8.04	74	100	74	75
5.20	4.68	7.28	100	37	27	68
5.17	4.54	9.99	100	100	49	67
5.14	4.42	5.87	50	100	75	34
5.17	3.72	6.44	100	53	20	3
			68. 4.99 to 4.90			
4.97	7.63	9.35	86	100	73	120
4.94	6.19	5.93	100	50	37	98
4.98	6.17	5.57	40	100	40	21
4.93	6.11	5.59	64	100	64	95
4.95	5.97	5.47	43	100	100	73
4.95	5.96	14.7	50	100	27	101
4.90	5.91	5.45	30	100	20	90
4.93	5.89	7.37	67	100	36	72
4.98	5.87	4.53	73	100	55	87
4.93	5.84	5.17	37	100	37	126
4.91	5.83	9.21	53	100	53	105
4.96	5.66	5.26	37	100	37	110
4.95	5.54	9.66	67	100	27	39

d(A)			I/I_1			Reference No.*
4.90	5.54	6.71	100	75	56	5
4.98	5.49	6.74	73	100	35	41
4.94	5.42	6.42	67	100	49	45
4.94	5.40	6.24	53	100	53	46
4.91	5.26	5.74	41	100	73	12
			67. 4.89 to 4.80			
4.82	8.04	5.01	100	74	74	75
4.87	7.03	6.19	100	49	36	17
4.82	6.24	5.39	75	100	50	84
4.86	6.07	5.10	40	100	53	19
4.85	5.99	6.66	100	82	67	89
4.85	5.97	5.72	100	100	100	82
4.80	5.91	5.11	47	100	39	53
4.81	5.91	4.61	58	100	36	18
4.89	5.89	7.20	86	100	55	107
4.85	5.84	9.66	73	100	27	79
4.89	5.70	4.21	53	100	53	51
4.87	5.68	2.89	100	87	38	112
4.82	5.54	5.85	100	100	40	38
4.89	5.45	8.04	83	100	49	115
4.85	4.45	5.85	100	73	55	137
			66. 4.79 to 4.70			
4.78	7.56	6.24	86	100	73	113
4.73	6.51	5.08	67	100	67	111
4.75	6.39	5.35	100	100	100	47
4.72	6.22	5.87	75	100	50	109
4.71	6.22	5.31	56	100	75	97
4.75	6.09	5.64	100	73	53	83
4.71	6.09	5.49	67	100	67	64
4.71	5.95	5.72	73	100	100	66
4.71	5.93	7.44	73	100	27	16
4.76	5.93	5.08	100	100	37	25
4.72	5.87	5.54	100	100	100	104
4.72	5.83	7.47	73	100	55	60
4.76	5.76	3.95	73	100	55	65
4.78	5.74	6.11	100	53	20	9
4.78	5.70	4.45	100	73	53	26
4.78	5.64	6.71	88	100	88	1
4.71	5.55	8.19	73	100	73	118
4.75	5.47	5.21	100	100	100	22
4.77	5.42	10.4	75	100	50	13
4.72	5.40	10.3	100	41	15	40
			65. 4.69 to 4.60			
4.67	6.24	5.02	94	100	100	62
4.62	6.13	5.68	73	100	73	26
4.68	6.07	5.55	73	100	73	52
4.69	5.97	5.47	73	100	73	88
4.61	5.91	4.81	36	100	58	18
4.64	5.87	5.31	40	100	100	130

d(A)				I/I_1			Reference No.*
4.66	5.81	11.2		73	100	56	91
4.65	5.79	11.2		50	100	50	86
4.62	5.55	14.5		100	73	53	32
4.65	5.55	5.01		73	100	100	28
4.66	5.49	10.5		53	100	53	7
4.68	5.20	7.28		37	100	27	68
			64. 4.59 to 4.50				
4.55	6.17	8.71		100	90	75	114
4.58	5.95	6.81		37	100	37	43
4.53	5.87	4.98		55	100	73	87
4.54	5.17	9.99		100	100	49	67
			63. 4.49 to 4.40				
4.42	6.26	6.76		53	100	53	69
4.42	5.87	5.14		100	75	50	34
4.42	5.85	5.70		37	100	75	94
4.42	5.54	15.0		20	100	20	125
4.46	5.37	10.4		37	100	27	99
4.45	4.85	5.85		73	100	55	137
4.45	4.78	5.70		53	100	73	26
			62. 4.39 to 4.30				
4.34	6.73	6.05		100	73	30	2
4.31	6.57	5.15		41	100	73	71
4.30	6.05	5.32		50	100	100	27
4.32	5.95	5.63		73	100	53	131
4.30	5.74	6.44		53	100	40	30
4.34	5.31	6.15		87	100	73	15
4.32	5.21	6.26		20	100	40	33
			61. 4.29 to 4.20				
4.27	6.13	5.49		100	27	27	56
4.24	6.01	5.16		56	100	75	48
4.22	5.74	5.44		90	100	100	77
4.21	5.70	4.89		53	100	53	51
4.24	5.57	11.8		40	100	27	37
4.20	5.33	5.59		36	100	67	92
4.24	5.26	6.42		56	100	75	134
			60. 4.19 to 4.10				
4.17	6.21	5.52		83	100	100	128
4.17	5.51	6.03		100	100	73	23
			58. 3.99 to 3.90				
3.95	5.76	4.76		55	100	73	65
			57. 3.89 to 3.80				
3.87	6.09	5.05		75	100	100	121

d(A)			I/I_1			Reference No.*
			56. 3.79 to 3.70			
3.74	5.47	6.89	37	100	37	42
3.72	5.17	6.44	53	100	20	3
			55. 3.69 to 3.60			
3.63	5.74	5.50	100	75	75	74
			52. 3.44 to 3.40			
3.44	5.79	5.07	73	100	40	6
			41. 2.89 to 2.85			
2.89	4.87	5.68	38	100	87	112

*Numbers in this column refer to the index of names given in Table I.

Table III.

1

d(A)	I/I_1
7.50	0.20
6.71	0.88
6.03	0.20
5.64	1.00
5.00	0.27
4.78	0.88
4.63	0.27
4.32	0.50
4.08	0.37
3.92	0.50
3.72	0.37
3.35	0.50
3.24	0.15
3.13	0.10
3.02	0.15
2.90	0.07
2.80	0.20
2.65	0.03
2.56	0.10
2.49	0.27
2.40	0.15
2.32	0.10
2.27	0.15
2.21	0.15
2.14	0.15
2.08	0.10
2.00	0.10
1.93	0.15
1.86	0.10
1.79	0.10

4

d(A)	I/I_1
12.0	0.15
7.80	0.15
7.06	0.37
5.85	1.00
5.41	0.27
5.14	0.75
4.71	0.37
4.11	0.37
3.79	0.27
3.46	0.27
3.04	0.15
2.55	0.15
2.48	0.20
2.41	0.20
2.28	0.10
2.18	0.20
1.97	0.20

2

d(A)	I/I_1
8.08	0.13
6.73	0.73
6.05	0.30
4.81	0.07
4.34	1.00
4.16	0.30
3.36	0.20
2.85	0.002
2.47	0.02
2.28	0.01
2.11	0.01
1.88	0.20

5

d(A)	I/I_1
8.68	0.03
8.12	0.38
6.71	0.56
6.35	0.05
6.01	0.05
5.54	0.75
5.35	0.08
4.90	1.00
4.50	0.08
4.30	0.08
4.05	0.38
3.87	0.56
3.68	0.15
3.38	0.28
3.25	0.28
3.14	0.03
3.08	0.20
2.98	0.20
2.89	0.11
2.76	0.01
2.63	0.15
2.53	0.11
2.46	0.05
2.40	0.20
2.35	0.15
2.30	0.15
2.23	0.05
2.15	0.20
2.09	0.08
2.03	0.08
1.96	0.15
1.93	0.11
1.86	0.15
1.79	0.20

3

d(A)	I/I_1
12.4	0.11
7.00	0.06
6.44	0.20
5.65	0.03
5.17	1.00
4.82	0.06
4.44	0.06
4.18	0.06
3.72	0.53
3.32	0.20
3.21	0.06
3.08	0.03
2.96	0.03
2.78	0.04
2.59	0.01
2.48	0.03
2.41	0.04
2.35	0.06
2.27	0.01
2.22	0.01
2.17	0.01
2.12	0.04
2.05	0.03
1.96	0.03
1.91	0.01
1.85	0.01
1.80	0.01
1.73	0.01

6

d(A)	I/I_1
9.16	0.06
7.31	0.04
6.52	0.03
5.79	1.00
5.07	0.40
4.60	0.11
4.40	0.20
4.06	0.11
3.82	0.01
3.64	0.03
3.44	0.73
3.33	0.20
3.13	0.04
2.97	0.15
2.87	0.06
2.71	0.11
2.60	0.11
2.50	0.11
2.41	0.06
2.31	0.08
2.27	0.08
2.22	0.11
2.18	0.08
2.15	0.11
2.11	0.04
2.04	0.08
1.95	0.06
1.92	0.08
1.87	0.06
1.82	0.04

7

d(A)	I/I_1
15.4	0.13
10.5	0.53
7.90	0.13
6.89	0.20
6.13	0.40
5.49	1.00
4.66	0.53
4.32	0.20
3.95	0.30
3.78	0.07
3.54	0.30
3.21	0.03
3.07	0.03
2.94	0.03
2.78	0.03
2.58	0.02
2.49	0.07
2.27	0.13
2.16	0.07
2.05	0.13
1.93	0.02

8

d(A)	I/I_1
12.8	0.05
9.87	0.05
7.53	0.41
6.54	0.73
5.83	1.00
5.54	0.55
5.02	0.73
4.76	0.55
4.29	0.27
4.01	0.20
3.79	0.08
3.65	0.15
3.44	0.05
3.32	0.05
3.19	0.20
3.08	0.05
3.00	0.05
2.73	0.20
2.63	0.11
2.53	0.08
2.45	0.08
2.36	0.02
2.21	0.20
2.14	0.08
2.09	0.11
1.98	0.08
1.90	0.08
1.81	0.02
1.77	0.02

9

d(A)	I/I_1
16.2	0.11
10.9	0.01
9.88	0.11
8.93	0.03
7.63	0.11
6.92	0.06
6.11	0.20
5.74	0.53
5.26	0.04
4.78	1.00
4.57	0.06
4.05	0.08
3.72	0.08
3.49	0.04
3.30	0.04
3.21	0.06
2.93	0.08
2.53	0.04
2.38	0.08
2.32	0.03
1.98	0.04

10

d(A)	I/I_1
17.0	0.30
13.6	0.17
11.3	0.23
9.76	0.04
8.76	0.08
7.31	0.11
6.71	0.23
6.21	0.30
5.59	0.50
5.17	1.00
4.76	0.17
4.52	0.11
4.33	0.11
4.10	0.17

11

d(A)	I/I_1
12.9	0.38
9.94	0.20
7.09	0.38
6.09	1.00
5.95	0.28
5.79	0.88
5.47	0.20
5.11	1.00
4.89	0.28
4.67	0.75
4.43	0.28
4.14	0.28
4.02	0.31
3.84	0.38
3.62	0.28
3.39	0.20
3.20	0.11
3.07	0.20
2.98	0.15
2.85	0.20
2.76	0.11
2.66	0.11
2.62	0.15
2.35	0.15
2.26	0.15
2.21	0.15
2.12	0.20
2.03	0.15
1.96	0.11
1.91	0.11

12

d(A)	I/I_1
17.0	0.15
13.4	0.20
11.4	0.15
9.26	0.15
8.43	0.24
7.76	0.11
6.86	0.20
6.26	0.20
5.74	0.73
5.26	1.00
4.91	0.41
4.58	0.20
4.22	0.20
3.97	0.05
3.80	0.20
3.58	0.11
3.41	0.15
3.19	0.04
2.94	0.04
2.66	0.02
2.42	0.01
2.27	0.05
2.22	0.02
2.14	0.15

13

d(A)	I/I_1
16.36	0.10
13.81	0.10
10.40	0.50
8.23	0.50
7.06	0.15
6.17	0.27
5.42	1.00
4.77	0.75
4.56	0.15
4.33	0.27
4.13	0.27
3.70	0.37
3.53	0.27
3.12	0.20
2.97	0.15
2.88	0.10
2.75	0.10
2.65	0.10
2.39	0.20
2.30	0.15
2.25	0.15
2.20	0.15
2.14	0.15
2.06	0.15
2.00	0.01
1.89	0.20
1.80	0.07
1.74	0.07

16

d(A)	I/I_1
7.44	0.27
6.49	0.15
5.93	1.00
4.71	0.73
4.53	0.27
4.00	0.27
3.85	0.15
3.62	0.11
3.47	0.11
3.28	0.09
2.92	0.11
2.71	0.05
2.64	0.05
2.44	0.08
2.36	0.08
2.28	0.08

14

d(A)	I/I_1
15.6	0.55
11.9	0.41
9.88	0.41
7.47	0.55
6.92	0.27
5.95	1.00
5.79	0.27
5.45	0.73
5.04	0.27
4.71	0.41
4.52	0.55
4.23	0.05
4.00	0.27
3.85	0.27
3.71	0.27
3.60	0.09
3.52	0.09
3.21	0.09
3.01	0.09
2.83	0.09
2.69	0.18
2.58	0.18
2.38	0.09
2.29	0.05
2.15	0.05
2.14	0.05

17

d(A)	I/I_1
16.5	0.04
10.2	0.04
9.21	0.20
7.60	0.04
7.03	0.49
6.19	0.36
5.75	0.13
5.44	0.13
5.10	0.13
4.87	1.00
4.66	0.20
4.30	0.13
4.12	0.13
3.98	0.13
3.87	0.13
3.78	0.13
3.74	0.27
3.59	0.13
3.38	0.13
3.26	0.13
3.10	0.13
3.02	0.01
2.94	0.01
2.78	0.01
2.57	0.13
2.50	0.13
2.39	0.13
2.34	0.13
2.28	0.20
2.21	0.13
2.14	0.01
2.09	0.20
2.00	0.01

15

d(A)	I/I_1
13.3	0.08
11.4	0.11
9.51	0.01
6.82	0.01
6.15	0.73
5.80	0.53
5.31	1.00
5.04	0.63
4.62	0.20
4.34	0.87
4.14	0.11
3.93	0.30
3.80	0.06
3.64	0.06
3.42	0.35
3.28	0.30
3.00	0.08
2.94	0.08
2.87	0.11
2.78	0.08
2.73	0.08
2.67	0.15
2.43	0.04
2.36	0.06
2.29	0.15
2.21	0.04
2.18	0.15
2.12	0.01
2.08	0.08
2.02	0.03

18

d(A)	I/I_1
13.2	0.02
9.36	0.02
7.41	0.20
7.00	0.09
5.91	1.00
4.81	0.58
4.61	0.36
4.05	0.27
3.48	0.20
3.27	0.09
2.92	0.02
2.83	0.02
2.43	0.02
2.28	0.13
1.19	0.09

19

d(A)	I/I_1
12.8	0.13
6.40	0.07
6.07	1.00
5.74	0.20
5.42	0.02
5.10	0.53
4.86	0.40
4.16	0.02
3.75	0.20
3.51	0.02
3.10	0.20
3.02	0.13
2.94	0.07
2.60	0.02
2.43	0.03
2.14	0.07
2.02	0.07
1.94	0.02

20

d(A)	I/I_1
12.6	0.06
11.1	0.01
8.76	0.11
7.76	0.04
6.53	0.30
6.12	0.40
5.56	1.00
5.25	0.53
4.37	0.40
4.12	0.08
3.88	0.08
3.54	0.25
3.27	0.11
3.08	0.03
3.00	0.08
2.93	0.08
2.78	0.08
2.57	0.08
2.48	0.06
2.40	0.04
2.31	0.08
2.23	0.08
2.08	0.03
2.03	0.08
1.98	0.03
1.94	0.04
1.85	0.03
1.78	0.04

21

d(A)	I/I_1
16.5	0.03
10.7	0.04
8.89	0.15
7.22	0.20
6.17	1.00
5.80	0.30
5.57	0.40
5.37	0.20
4.98	0.40
4.56	0.20
4.35	0.15
4.20	0.11
3.86	0.30
3.61	0.08
3.36	0.04
3.04	0.11
2.90	0.08
2.81	0.11
2.67	0.01
2.61	0.04
2.52	0.06
2.44	0.08
2.30	0.04
2.18	0.06
2.10	0.06
2.07	0.01
1.97	0.03
1.86	0.01
1.75	0.04

22

d(A)	I/I_1
14.3	0.05
12.5	0.05
8.35	0.08
7.44	0.11
6.17	0.11
5.82	0.08
5.47	1.00
5.21	1.00
4.90	0.08
4.75	1.00
4.25	0.55
3.91	0.11
3.75	0.11
3.62	0.05
3.35	0.27
3.09	0.20
3.01	0.20
2.92	0.11
2.73	0.08
2.49	0.11
2.34	0.03
2.25	0.05
2.17	0.05
2.03	0.08
1.80	0.08

23

d(A)	I/I_1
12.3	0.08
8.27	0.15
6.61	0.06
6.03	0.73
5.51	1.00
4.98	0.40
4.58	0.25
4.17	1.00
3.96	0.30
3.76	0.17
3.60	0.17
3.41	0.06
3.25	0.46
3.14	0.11
3.01	0.25
2.92	0.04
2.83	0.11
2.75	0.15
2.68	0.11
2.59	0.20
2.49	0.08
2.40	0.34
2.31	0.04
2.26	0.04
2.20	0.04
2.14	0.06
2.11	0.11
2.07	0.06
2.04	0.06

24

d(A)	I/I_1
10.8	0.15
8.47	0.27
7.09	0.02
6.05	0.41
5.87	1.00
5.58	1.00
5.39	0.64
5.00	0.34
4.82	0.41
4.50	0.17
4.28	0.04
4.09	0.24
3.85	0.11
3.72	0.15
3.60	0.04
3.46	0.05
3.38	0.27
3.27	0.11
3.11	0.08
3.02	0.11
2.94	0.27
2.82	0.11
2.71	0.15
2.58	0.08
2.56	0.04
2.48	0.04
2.38	0.08
2.31	0.15
2.10	0.15
2.05	0.11
1.98	0.11
1.94	0.05

25

d(A)	I/I_1
14.7	0.27
7.40	0.27
6.89	0.03
5.93	1.00
5.55	0.15
5.08	0.37
4.76	1.00
4.59	0.37
4.20	0.20
4.02	0.27
3.76	0.10
3.64	0.15
3.51	0.20
3.42	0.15
3.32	0.20
3.20	0.10
2.92	0.15
2.84	0.10
2.74	0.10
2.64	0.15
2.54	0.10
2.40	0.15
2.32	0.15
2.30	0.15
2.13	0.03
2.02	0.10
1.98	0.07
1.91	0.10
1.86	0.03

27

d(A)	I/I_1
14.9	0.07
8.31	0.10
7.38	0.07
6.05	1.00
5.59	0.28
5.32	1.00
5.02	0.02
4.67	0.28
4.50	0.20
4.30	0.50
3.92	0.27
3.69	0.27
3.59	0.27
3.47	0.15
3.35	0.27
3.23	0.03
3.14	0.15
3.05	0.10
2.92	0.10
2.83	0.07
2.78	0.07
2.72	0.15
2.59	0.02
2.50	0.02
2.39	0.07
2.32	0.20
2.22	0.03
2.13	0.10
2.09	0.10
2.03	0.10
1.96	0.01

26

d(A)	I/I_1
16.7	0.08
9.07	0.01
8.30	0.11
7.31	0.15
5.70	0.73
5.25	0.11
4.78	1.00
4.45	0.53
4.43	0.01
4.04	0.15
3.77	0.06
3.47	0.11
3.23	0.08
3.11	0.08
2.94	0.11
2.84	0.04
2.79	0.04
2.59	0.01
2.41	0.06
2.34	0.06
2.28	0.08
2.24	0.06
2.19	0.04
2.13	0.04
2.09	0.04
2.05	0.04
2.01	0.04
1.95	0.11
1.87	0.01
1.85	0.08
1.76	0.03
1.74	0.03

28

d(A)	I/I_1
12.1	0.30
10.2	0.40
8.47	0.30
7.56	0.07
7.05	0.40
6.05	0.53
5.55	1.00
5.31	0.20
5.01	1.00
4.65	0.73
4.32	0.53
3.90	0.53
3.63	0.73
3.50	0.07
3.36	0.40
3.06	0.53
2.87	0.40
2.78	0.07
2.71	0.30
2.53	0.13
2.38	0.40
2.34	0.40
2.25	0.53
2.16	0.40
2.11	0.40
2.06	0.40
2.00	0.20
1.97	0.20
1.91	0.30
1.85	0.03

29

d(A)	I/I_1
12.0	0.08
10.0	0.03
8.98	0.28
7.14	0.20
6.42	0.20
6.07	1.00
5.75	0.38
5.38	0.56
5.13	0.11
4.91	0.20
4.51	0.11
4.17	0.15
4.04	0.08
3.75	0.15
3.53	0.05
3.36	0.08
3.26	0.08
3.12	0.08
2.93	0.08
2.80	0.11
2.62	0.08
2.49	0.05
2.39	0.05
2.15	0.08
2.09	0.07

30

d(A)	I/I_1
12.1	0.06
10.6	0.04
8.51	0.15
6.44	0.40
5.74	1.00
5.21	0.20
4.76	0.25
4.52	0.13
4.30	0.53
4.12	0.30
3.86	0.18
3.75	0.11
3.56	0.03
3.36	0.06
3.24	0.20
3.10	0.15
2.94	0.11
2.86	0.04
2.79	0.20
2.67	0.15
2.60	0.08
2.49	0.10
2.41	0.08
2.35	0.10
2.26	0.08
2.20	0.08
2.15	0.15
2.12	0.13
2.08	0.13
2.02	0.08
1.95	0.04
1.89	0.01
1.86	0.11

31

d(A)	I/I_1
12.1	0.13
10.5	0.13
9.46	0.03
8.47	0.07
7.09	0.30
6.37	0.53
5.75	1.00
5.38	0.53
4.85	0.40
4.53	0.30
4.32	0.40
4.15	0.30
3.78	0.20
3.56	0.30
3.29	0.30
3.11	0.03
2.99	0.03
2.84	0.40
2.70	0.02
2.53	0.02
2.43	0.03
2.16	0.02
2.08	0.03

32

d(A)	I/I_1
14.5	0.53
10.3	0.13
8.85	0.07
8.04	0.30
7.53	0.40
6.84	0.30
5.85	0.40
5.55	0.73
5.14	0.30
4.62	1.00
4.20	0.07
3.97	0.53
3.81	0.13
3.65	0.13
3.47	0.30
3.37	0.40
3.22	0.13
3.05	0.40
2.89	0.53
2.75	0.07
2.62	0.02
2.56	0.40
2.41	0.13
2.34	0.13
2.31	0.40
2.21	0.40
2.03	0.07
1.98	0.07
1.87	0.07
1.81	0.13
1.60	0.02
1.52	0.02

33

d(A)	I/I_1
15.6	0.06
12.5	0.08
10.8	0.11
8.98	0.01
7.73	0.08
7.03	0.11
6.26	0.40
5.85	0.04
5.29	0.04
5.21	1.00
4.76	0.11
4.39	0.04
4.32	0.20
3.85	0.08
3.68	0.15
3.54	0.04
3.38	0.04
3.24	0.03
3.15	0.01
2.83	0.01
2.70	0.02
2.55	0.02
2.37	0.01
2.32	0.03
2.23	0.02
2.17	0.03

34

d(A)	I/I_1
6.19	0.32
5.87	0.75
5.14	0.50
4.85	0.43
4.42	1.00
4.19	0.27
3.85	0.32
3.70	0.20
3.49	0.28
3.15	0.32
2.94	0.27
2.76	0.15
2.56	0.27
2.50	0.07
2.42	0.10
2.36	0.15
2.18	0.20
2.01	0.20
1.88	0.07
1.82	0.07

35

d(A)	I/I_1
11.6	0.55
10.5	0.05
9.07	0.27
8.39	0.55
7.17	0.55
6.44	0.55
5.83	0.73
5.32	1.00
4.87	0.41
4.49	0.41
4.14	0.41
3.92	0.41
3.69	0.41
3.68	0.05
2.71	0.02
2.52	0.02
2.35	0.02
2.21	0.02

36

d(A)	I/I_1
11.7	0.15
9.87	0.10
8.19	0.07
6.89	0.03
6.19	1.00
5.78	0.03
5.47	0.50
5.25	0.37
4.98	0.07
4.64	0.20
4.49	0.27
4.36	0.20
4.20	0.15
3.87	0.20
3.79	0.03
3.43	0.37
3.25	0.03
3.09	0.27
2.84	0.10
2.75	0.20
2.66	0.20
2.58	0.15
2.48	0.10
2.40	0.10
2.35	0.10
2.28	0.20
2.18	0.10
2.14	0.10
2.05	0.15
2.00	0.15
1.93	0.20

37

d(A)	I/I_1
11.8	0.27
10.4	0.04
8.31	0.01
6.21	0.27
5.57	1.00
5.08	0.11
4.65	0.08
4.24	0.40
4.06	0.15
3.78	0.05
3.65	0.15
3.38	0.04
3.28	0.20
3.18	0.11
3.08	0.08
2.89	0.11
2.72	0.04
2.65	0.05
2.55	0.02
2.39	0.05
2.29	0.08
2.19	0.11
2.12	0.05
2.08	0.05
2.03	0.04
1.86	0.01
1.80	0.02
1.64	0.01

38

d(A)	I/I_1
9.31	0.15
6.24	0.30
5.85	0.40
5.54	1.00
5.23	0.20
4.82	1.00
4.67	0.08
4.11	0.08
3.84	0.04
3.51	0.15
3.35	0.20
3.19	0.30
3.00	0.15
2.94	0.06
2.85	0.08
2.77	0.08
2.59	0.08
2.50	0.04
2.36	0.11
2.32	0.11
2.24	0.06
2.15	0.06
2.08	0.08
2.05	0.08
1.98	0.04
1.94	0.04
1.88	0.06
1.79	0.04
1.77	0.04

39

d(A)	I/I_1
16.7	0.13
12.7	0.13
11.3	0.01
9.66	0.27
8.59	0.09
7.87	0.13
7.34	0.13
6.49	0.13
5.54	1.00
4.95	0.67
4.56	0.27
4.21	0.20
3.92	0.20
3.68	0.13
3.51	0.13
3.37	0.20
3.23	0.20
3.08	0.01
3.00	0.01
2.90	0.01
2.54	0.13
2.45	0.04
2.38	0.04
2.30	0.04
2.22	0.02
2.09	0.01
1.99	0.01

40

d(A)	I/I_1
10.3	0.15
9.45	0.15
8.08	0.11
7.09	0.02
6.22	0.08
5.91	0.11
5.66	0.09
5.40	0.41
4.72	1.00
4.34	0.08
4.16	0.08
4.01	0.11
3.74	0.08
3.51	0.10
3.31	0.08
3.14	0.11
3.00	0.09
2.90	0.09
2.60	0.02
2.46	0.02
2.32	0.05
2.27	0.04
2.13	0.05
2.09	0.05
1.98	0.02
1.89	0.04

41

d(A)	I/I_1
12.8	0.08
9.56	0.30
8.72	0.11
7.87	0.11
6.74	0.35
6.03	0.01
5.49	1.00
4.98	0.73
4.70	0.30
4.30	0.30
3.97	0.08
3.64	0.20
3.38	0.01
3.22	0.15
3.10	0.11
2.93	0.04
2.77	0.03
2.66	0.03
2.53	0.15
2.46	0.06
2.39	0.01
2.31	0.04
2.25	0.04

42

d(A)	I/I_1
13.7	0.20
10.5	0.27
7.63	0.02
6.89	0.37
6.30	0.27
5.47	1.00
4.80	0.03
4.43	0.27
4.11	0.20
3.93	0.20
3.74	0.37
3.48	0.07
3.31	0.07
3.13	0.20
2.89	0.10
2.77	0.01
2.65	0.15
2.35	0.15
2.31	0.20
2.20	0.20
2.12	0.20
2.05	0.03
1.96	0.07
1.86	0.01
1.82	0.07
1.77	0.07
1.72	0.03
1.68	0.03

43

d(A)	I/I_1
11.0	0.27
8.43	0.20
6.81	0.37
5.95	1.00
5.25	0.27
4.98	0.27
4.58	0.37
4.22	0.20
4.06	0.20
3.85	0.15
3.69	0.10
3.48	0.10
3.33	0.10
3.22	0.10
3.11	0.03
2.99	0.15
2.88	0.10
2.81	0.03
2.62	0.10
2.50	0.03
2.44	0.03
2.37	0.07
2.28	0.02
2.20	0.03
2.10	0.02
1.93	0.03
1.89	0.03

44

d(A)	I/I_1
10.9	0.04
6.09	0.40
5.91	0.30
5.59	1.00
5.11	0.30
4.54	0.03
4.34	0.08
4.12	0.08
3.72	0.04
3.58	0.04
3.42	0.03
3.29	0.04
3.21	0.20
3.11	0.15
3.01	0.06
2.87	0.08
2.76	0.06
2.67	0.06
2.58	0.08
2.48	0.01
2.37	0.06
2.28	0.03
2.17	0.04
2.10	0.04
2.03	0.01
1.96	0.06
1.80	0.01
1.68	0.01

45

d(A)	I/I_1
10.9	0.13
9.66	0.02
6.42	0.49
6.03	0.27
5.42	1.00
4.94	0.67
4.70	0.20
4.30	0.13
3.87	0.13
3.60	0.13
3.47	0.09
3.37	0.09
3.29	0.09
3.06	0.09
2.98	0.20
2.86	0.36
2.77	0.04
2.69	0.04
2.58	0.36
2.52	0.13
2.34	0.13
2.32	0.20
2.25	0.04
2.17	0.13
2.14	0.13
2.03	0.09
2.00	0.09
1.91	0.13
1.88	0.13
1.84	0.02
1.80	0.01

46

d(A)	I/I_1
11.2	0.30
9.02	0.01
7.90	0.03
6.86	0.01
6.24	0.53
5.40	1.00
4.94	0.53
4.48	0.08
4.14	0.30
3.87	0.15
3.74	0.11
3.56	0.30
3.32	0.08
3.22	0.20
3.04	0.25
3.82	0.11
2.69	0.06
2.62	0.06
2.55	0.06
2.44	0.04
2.35	0.04
2.28	0.11
2.24	0.15
2.19	0.03
2.12	0.06
2.04	0.08
1.97	0.03
1.95	0.04
1.89	0.04
1.83	0.04

47

d(A)	I/I_1
13.8	0.28
7.53	0.05
6.94	0.11
6.39	1.00
5.88	0.01
5.35	1.00
4.93	0.38
4.75	1.00
4.55	0.20
4.24	0.38
3.83	0.15
3.65	0.08
3.55	0.11
3.46	0.11
3.27	0.20
3.06	0.56
2.90	0.56
2.81	0.03
2.67	0.15
2.51	0.20
2.43	0.15
2.24	0.03
2.19	0.03
2.12	0.11
2.03	0.03
1.99	0.05
1.92	0.11
1.79	0.08
1.75	0.08

48

d(A)	I/I_1
10.4	0.01
8.51	0.01
7.09	0.33
6.71	0.03
6.01	1.00
5.57	0.33
5.16	0.75
4.68	0.05
4.45	0.15
4.24	0.56
4.00	0.20
3.66	0.18
3.53	0.28
3.19	0.20
3.13	0.08
3.05	0.08
2.98	0.11
2.80	0.15
2.69	0.01
2.51	0.15
2.43	0.15
2.38	0.03
2.31	0.03
2.24	0.08
2.12	0.15
2.08	0.03
2.03	0.08
2.00	0.08
1.95	0.08

49

d(A)	I/I_1
10.3	0.20
6.56	0.20
6.28	1.00
5.79	0.73
5.56	0.73
5.11	0.53
4.46	0.53
3.91	0.20
3.53	0.40
3.40	0.13
3.13	0.07
2.97	0.13
2.88	0.07
2.77	0.07
2.67	0.07
2.53	0.03
2.43	0.07
2.39	0.03
2.23	0.07
2.16	0.13
2.10	0.03
2.01	0.03
1.96	0.13

50

9.33	0.07
8.33	0.50
6.96	0.63
6.12	0.37
5.73	0.37
5.24	1.00
4.83	0.20
4.64	0.10
4.51	0.07
4.19	0.15
3.95	0.37
3.71	0.07
3.46	0.27
3.36	0.37
3.24	0.02
3.16	0.03
3.08	0.02
3.01	0.10
2.90	0.03
2.78	0.10
2.69	0.10
2.60	0.03
2.39	0.07
2.32	0.10
2.26	0.10
2.22	0.03
2.16	0.03
2.11	0.03
2.04	0.10
1.97	0.07
1.86	0.07
1.83	0.10

51

d(A)	I/I_1
8.59	0.30
6.66	0.30
6.33	0.40
5.70	1.00
5.49	0.30
5.15	0.02
4.89	0.53
4.65	0.02
4.44	0.02
4.21	0.53
4.10	0.30
3.80	0.02
3.68	0.02
3.49	0.40
3.29	0.13
3.19	0.03
2.99	0.03
2.77	0.03
2.44	0.30
2.38	0.30
2.25	0.07
2.18	0.30

52

d(A)	I/I_1
13.3	0.20
10.3	0.11
7.94	0.06
7.34	0.11
6.74	0.11
6.07	1.00
5.55	0.73
4.94	0.30
4.68	0.73
4.39	0.30
4.16	0.30
3.95	0.20
3.82	0.20
3.69	0.08
3.54	0.08
3.30	0.20
3.16	0.08
2.97	0.06
2.80	0.06
2.63	0.08
2.56	0.08
2.46	0.15
2.36	0.04
2.33	0.03
2.26	0.08
2.18	0.08
2.00	0.06
1.91	0.06
1.88	0.01
1.83	0.01
1.76	0.01

53

d(A)	I/I_1
11.1	0.13
8.67	0.13
7.38	0.04
6.81	0.13
6.46	0.04
5.91	1.00
5.11	0.39
4.80	0.47
4.63	0.23
4.52	0.23
4.28	0.17
4.12	0.17
3.94	0.09
3.79	0.06
3.63	0.17
3.51	0.04
3.30	0.04
3.19	0.17
3.06	0.13
2.96	0.13
2.84	0.13
2.70	0.08
2.54	0.02
2.48	0.04
2.42	0.04
2.33	0.17
2.25	0.06
2.20	0.06
2.15	0.17
1.97	0.13
1.91	0.13
1.79	0.02

54

d(A)	I/I_1
10.4	0.38
6.79	0.25
6.01	1.00
5.74	1.00
5.42	0.25
5.16	0.56
4.80	0.56
4.51	0.38
4.19	0.25
4.10	0.25
3.79	0.03
3.55	0.25
3.38	0.25
3.28	0.25
3.11	0.56
3.02	0.06
2.93	0.13
2.82	0.13
2.67	0.06
2.57	0.03
2.48	0.06
2.38	0.25
2.25	0.13
2.09	0.06
2.03	0.25
1.94	0.03
1.86	0.25
1.78	0.03
1.72	0.03
1.63	0.06

55

d(A)	I/I_1
7.94	0.04
6.86	0.11
6.13	1.00
5.68	0.73
5.26	0.20
4.62	0.73
4.42	0.30
4.03	0.25
3.82	0.25
3.70	0.20
3.13	0.30
3.01	0.27
2.90	0.15
2.77	0.06
2.70	0.06
2.59	0.08
2.45	0.08
2.34	0.11
2.26	0.11
2.21	0.15
2.10	0.08
1.99	0.03
1.85	0.08
1.82	0.03
1.75	0.03
1.71	0.06

56

d(A)	I/I_1
15.0	0.20
9.98	0.04
7.70	0.04
6.13	0.27
5.49	0.27
5.14	0.20
4.27	1.00
3.75	0.08
3.48	0.01
3.08	0.08
2.74	0.15
2.51	0.08
2.37	0.04
2.21	0.03
2.05	0.03

57

d(A)	I/I_1
11.3	0.07
9.07	0.53
8.35	0.13
6.54	0.30
6.11	0.30
5.77	0.40
5.41	1.00
5.11	0.53
4.72	0.53
4.50	0.40
4.22	0.40
3.90	0.30
3.67	0.30
3.44	0.40
3.28	0.13
3.11	0.20
3.01	0.20
2.81	0.13
2.77	0.13
2.69	0.13
2.54	0.13
2.44	0.13
2.34	0.13
2.31	0.13
2.13	0.30
2.05	0.20
1.99	0.20
1.90	0.20
1.84	0.20
1.81	0.13
1.75	0.03
1.68	0.02
1.55	0.02
1.49	0.02
1.36	0.03

58

d(A)	I/I_1
12.1	0.15
10.8	0.15
6.49	1.00
5.99	0.40
5.49	0.87
5.02	1.00
4.65	0.20
4.09	0.23
3.87	0.17
3.73	0.15
3.60	0.15
3.49	0.20
3.28	0.11
3.09	0.35
3.01	0.30
2.90	0.06
2.78	0.23
2.62	0.20
2.41	0.15
2.34	0.11
2.30	0.11
2.22	0.11
2.17	0.15
2.12	0.04
2.08	0.15
2.02	0.08
1.98	0.08
1.91	0.15

59

d(A)	I/I_1
11.9	0.20
7.63	0.37
6.78	0.75
6.01	1.00
5.54	1.00
5.20	0.88
4.61	0.37
4.41	0.63
4.12	0.63
4.01	0.63
3.87	0.20
3.70	0.27
3.58	0.27
3.25	0.37
2.98	0.37
2.80	0.10
2.68	0.37
2.51	0.27
2.43	0.10
2.34	0.15
2.31	0.57
2.24	0.10
2.15	0.10
2.07	0.20
1.99	0.03
1.95	0.10

60

d(A)	I/I_1
14.2	0.27
8.47	0.05
7.47	0.55
5.83	1.00
5.32	0.27
4.88	0.15
4.72	0.73
4.52	0.27
4.33	0.05
3.93	0.41
3.71	0.20
3.51	0.27
3.35	0.11
3.25	0.20
2.90	0.20
2.78	0.20
2.60	0.15
2.45	0.04
2.35	0.15
2.24	0.15
2.16	0.06
2.09	0.05
2.01	0.11
1.96	0.11
1.82	0.04
1.64	0.02

61		62		63	
d(A)	I/I_1	d(A)	I/I_1	d(A)	I/I_1
13.9	0.38	6.24	1.00	12.3	0.15
10.5	0.56	5.95	0.56	10.8	0.15
8.04	0.56	5.40	0.47	9.56	0.15
6.89	0.38	5.02	1.00	7.06	0.15
6.46	0.25	4.67	0.94	6.09	0.75
6.08	1.00	4.16	0.15	5.87	0.20
5.66	1.00	4.02	0.28	5.64	1.00
5.23	1.00	3.85	0.11	5.31	0.88
4.95	0.56	3.67	0.15	5.06	0.27
4.51	0.56	3.50	0.28	4.76	0.20
4.42	0.56	3.41	0.08	4.48	0.43
4.12	0.38	3.28	0.05	4.18	0.37
3.94	0.25	3.13	0.20	3.82	0.15
3.79	0.38	3.02	0.20	3.67	0.27
3.68	0.25	2.86	0.20	3.46	0.15
3.53	0.38	2.71	0.15	3.24	0.15
3.36	0.38	2.51	0.15	3.06	0.15
3.20	0.38	2.46	0.15	2.98	0.15
3.08	0.38	2.42	0.11	2.86	0.10
2.92	0.03	2.34	0.03	2.75	0.03
2.81	0.56	2.19	0.11	2.53	0.20
2.61	0.13	2.11	0.03	2.43	0.03
2.45	0.38	2.07	0.03	2.38	0.03
2.37	0.13	2.05	0.05	2.25	0.03
2.33	0.06	2.00	0.11	2.21	0.03
2.27	0.13	1.96	0.11	2.04	0.03
2.11	0.25	1.93	0.08	1.99	0.03
2.05	0.25	1.83	0.05	1.85	0.03
1.99	0.38	1.81	0.05	1.78	0.03
1.89	0.13	1.75	0.05		
		1.62	0.04		
		1.51	0.03		

64

d(A)	I/I_1
10.8	0.36
7.94	0.27
7.14	0.36
6.09	1.00
5.49	0.67
5.08	0.13
4.84	0.04
4.71	0.67
4.40	0.27
4.22	0.04
3.97	0.27
3.87	0.27
3.73	0.01
3.60	0.27
3.45	0.27
3.24	0.09
3.11	0.09
2.95	0.27
2.87	0.13
2.80	0.13
2.73	0.09
2.68	0.09
2.60	0.09
2.55	0.09
2.50	0.02
2.45	0.04
2.41	0.04
2.37	0.04
2.28	0.27
2.22	0.02
2.15	0.27
2.07	0.04
2.01	0.02
1.98	0.02
1.94	0.02

65

d(A)	I/I_1
14.3	0.05
7.97	0.27
7.14	0.11
6.11	0.27
5.76	1.00
5.55	0.08
4.76	0.73
4.53	0.27
4.32	0.02
4.04	0.20
3.95	0.55
3.76	0.08
3.63	0.15
3.51	0.15
3.37	0.11
3.20	0.04
3.09	0.02
2.91	0.11
2.77	0.15
2.65	0.11
2.56	0.04
2.49	0.08
2.37	0.08
2.26	0.20
2.18	0.02
2.05	0.05
1.99	0.05
1.90	0.04
1.84	0.04
1.75	0.01
1.71	0.02
1.70	0.02

66

d(A)	I/I_1
10.9	0.40
7.53	0.40
6.78	0.40
5.95	1.00
5.72	1.00
5.29	0.53
5.00	0.63
4.71	0.73
4.22	0.25
3.94	0.13
3.60	0.13
3.33	0.25
3.05	0.53
2.82	0.20
2.51	0.13
2.20	0.13

67

d(A)	I/I_1
9.99	0.49
8.31	0.09
6.73	0.49
6.05	0.09
5.49	0.09
5.17	1.00
4.99	0.04
4.70	0.04
4.54	1.00
4.41	0.20
4.12	0.20
3.72	0.27
3.58	0.09
2.95	0.13
2.74	0.09
2.62	0.09
2.47	0.09
2.28	0.11
2.14	0.02

68

d(A)	I/I_1
14.7	0.10
10.4	0.20
7.28	0.27
5.59	0.20
5.20	1.00
4.68	0.37
4.37	0.03
4.20	0.03
3.92	0.20
3.56	0.20
3.43	0.10
3.31	0.10
3.16	0.15
3.00	0.15
2.90	0.03
2.78	0.20
2.50	0.03
2.41	0.15
2.34	0.15
2.27	0.03
2.17	0.20
2.11	0.02
2.04	0.20
1.96	0.07
1.91	0.07
1.88	0.02
1.76	0.07
1.69	0.07

69

d(A)	I/I_1
11.2	0.11
9.35	0.20
7.83	0.01
6.76	0.53
6.26	1.00
5.74	0.11
5.20	0.20
4.66	0.20
4.42	0.53
4.23	0.11
4.08	0.11
3.88	0.08
3.70	0.08
3.58	0.11
3.39	0.15
3.28	0.11
3.09	0.15
2.92	0.11
2.78	0.01
2.69	0.08
2.55	0.08
2.41	0.11
2.32	0.01
2.22	0.15
2.10	0.01
2.07	0.01
2.01	0.08
1.91	0.15
1.82	0.01
1.79	0.01

70

d(A)	I/I_1
15.1	0.09
8.08	0.09
7.37	0.27
6.59	0.37
6.01	1.00
5.80	0.49
5.54	0.49
5.18	0.27
4.88	0.49
4.40	0.36
4.12	0.49
3.90	0.27
3.75	0.13
3.63	0.36
3.37	0.09
3.13	0.09
2.97	0.04
2.91	0.13
2.63	0.27
2.50	0.04
2.43	0.04
2.34	0.13
2.33	0.04
2.18	0.09
2.11	0.09
2.00	0.20
1.94	0.04
1.88	0.04

71

d(A)	I/I_1
9.51	0.15
7.22	0.05
6.57	1.00
5.78	0.08
5.51	0.21
5.15	0.73
4.84	0.08
4.54	0.08
4.31	0.41
4.03	0.11
3.92	0.11
3.68	0.15
3.59	0.15
3.43	0.20
3.29	0.15
3.23	0.11
2.99	0.15
2.88	0.11
2.78	0.05
2.66	0.11
2.57	0.15
2.41	0.11
2.36	0.11
2.28	0.11
2.19	0.08
2.16	0.11
2.11	0.15
2.02	0.20
1.95	0.08
1.87	0.02
1.81	0.04
1.78	0.05

72

d(A)	I/I_1
13.2	0.27
10.0	0.09
8.15	0.09
7.37	0.36
5.89	1.00
4.93	0.67
4.70	0.27
4.53	0.04
4.08	0.20
3.80	0.20
3.62	0.09
3.42	0.04
3.32	0.09
3.09	0.02
3.00	0.20
2.68	0.02
2.48	0.04
2.44	0.13
2.31	0.04
2.22	0.13
2.07	0.09

73

d(A)	I/I_1
12.1	0.16
8.47	0.16
5.97	1.00
5.47	1.00
4.95	0.43
4.30	0.16
4.02	0.43
3.83	0.24
3.47	0.05
3.35	0.05
3.20	0.43
2.92	0.16
2.83	0.16
2.74	0.05
2.66	0.05
2.59	0.05
2.37	0.11
2.23	0.16
1.99	0.32
1.87	0.05
1.80	0.05
1.72	0.05

74

d(A)	I/I_1
8.15	0.27
7.66	0.10
7.00	0.63
6.61	0.07
6.28	0.50
6.06	0.50
5.74	0.75
5.50	0.75
5.16	0.15
4.75	0.75
4.47	0.37
4.26	0.03
4.02	0.20
3.82	0.20
3.63	1.00
3.41	0.20
3.29	0.15
3.21	0.15
3.11	0.20
2.97	0.10
2.84	0.15
2.71	0.15
2.66	0.03
2.54	0.07
2.48	0.07
2.41	0.07
2.29	0.20
2.23	0.15
2.17	0.10
2.07	0.07
2.04	0.03
2.01	0.10
1.96	0.20
1.88	0.15
1.84	0.15

75

d(A)	I/I_1
13.1	0.04
8.04	0.74
7.53	0.32
7.09	0.32
5.79	0.63
5.01	0.74
4.82	1.00
4.68	0.23
4.16	0.23
3.87	0.17
3.70	0.39
3.54	0.06
3.41	0.13
3.30	0.13
3.23	0.32
3.00	0.23
2.94	0.23
2.85	0.09
2.81	0.09
2.71	0.04
2.62	0.09
2.50	0.09
2.41	0.13
2.35	0.13
2.28	0.13
2.21	0.09
2.19	0.09
2.12	0.04
2.08	0.04
2.02	0.17
1.95	0.01
1.90	0.09
1.85	0.04
1.77	0.04

76

d(A)	I/I_1
11.9	0.16
8.39	0.24
6.49	0.05
6 07	1.00
5.74	1.00
5.41	0.32
5.10	0.59
4.71	0.24
4.43	0.24
4.22	0.05
3.92	0.24
3.82	0.16
3.70	0.16
3.43	0.16
3.30	0.24
3.18	0.24
3.05	0.16
2.92	0.05
2.74	0.11
2.71	0.16
2.63	0.16
2.50	0.03
2.39	0.32
2.25	0.11
2.19	0.11
2.10	0.11
2.00	0.16
1.96	0.11
1.80	0.03

77

d(A)	I/I_1
12.5	0.42
10.6	0.13
8.12	0.04
6.13	0.27
5.74	1.00
5.44	1.00
5.01	0.20
4.83	0.09
4.22	0.67
4.10	0.13
3.93	0.04
3.81	0.09
3.47	0.09
3.30	0.36
3.18	0.02
3.08	0.20
2.97	0.20
2.73	0.09
2.02	0.09
1.88	0.09

78

d(A)	I/I_1
19.0	0.55
11.6	0.41
9.36	0.05
6.89	0.09
6.33	0.73
6.17	1.00
5.77	0.73
5.50	0.73
5.35	0.73
5.05	0.73
4.67	0.55
4.52	0.09
4.33	0.73
4.13	0.18
3.94	0.55
3.85	0.09
3.69	0.18
3.58	0.09
3.49	0.09
3.41	0.55
3.34	0.41
3.16	0.09
3.07	0.09
3.01	0.09
2.88	0.09
2.77	0.09
2.73	0.09
2.66	0.18
2.59	0.02
2.50	0.02
2.43	0.02
2.36	0.09
2.31	0.18
2.28	0.18
2.23	0.18
2.17	0.18
2.13	0.02
2.07	0.09
2.01	0.09

79

d(A)	I/I_1
9.66	0.27
8.94	0.24
6.61	0.05
6.37	0.08
5.84	1.00
5.17	0.08
4.85	0.73
4.27	0.15
4.11	0.15
3.96	0.15
3.83	0.04
3.65	0.11
3.52	0.04
3.29	0.20
2.99	0.20
2.82	0.04
2.69	0.15
2.59	0.08
2.54	0.11
2.47	0.04
2.34	0.04
2.29	0.02
2.22	0.11
2.17	0.02
2.10	0.05
2.05	0.05
1.98	0.15
1.88	0.08
1.81	0.02
1.75	0.02
1.67	0.05

80

d(A)	I/I_1
10.5	0.06
6.84	0.15
6.40	0.04
5.80	1.00
5.61	0.03
5.28	0.20
5.14	0.15
4.76	0.15
4.46	0.08
4.18	0.08
3.86	0.11
3.65	0.15
3.43	0.15
3.20	0.06
2.94	0.06
2.82	0.06
2.72	0.08
2.62	0.08
2.54	0.11
2.36	0.04
2.17	0.04
2.14	0.06
2.02	0.03
1.92	0.03
1.89	0.03

81

d(A)	I/I_1
12.1	0.27
6.01	1.00
5.83	1.00
5.06	0.75
4.85	0.27
4.62	0.27
4.30	0.75
4.08	0.20
3.75	0.10
3.62	0.20
3.52	0.10
3.40	0.15
3.31	0.15
3.17	0.15
3.08	0.15
3.00	0.27
2.92	0.15
2.86	0.15
2.82	0.15
2.74	0.10
2.68	0.15
2.57	0.03
2.47	0.03
2.42	0.03
2.38	0.07
2.36	0.03
2.30	0.07
2.28	0.03
2.21	0.03
2.13	0.01
2.08	0.20
2.04	0.20
2.01	0.01
1.99	0.20
1.95	0.20
1.92	0.01
1.90	0.01
1.88	0.01

82

d(A)	I/I_1
9.77	0.27
9.03	0.27
6.66	0.09
6.37	0.09
5.97	1.00
5.72	1.00
5.17	0.09
4.85	1.00
4.28	0.17
4.15	0.17
3.97	0.17
3.66	0.17
3.51	0.04
3.29	0.20
3.00	0.20
2.82	0.02
2.69	0.13
2.56	0.13
2.38	0.04
2.36	0.04
2.20	0.13
2.09	0.04
1.98	0.13
1.89	9 0.04

83

d(A)	I/I_1
15.5	0.13
7.83	0.07
6.61	0.02
6.09	0.73
5.64	0.53
5.33	0.30
5.19	0.30
5.00	0.13
4.75	1.00
4.45	0.13
4.28	0.07
3.90	0.40
3.76	0.40
3.56	0.40
3.40	0.07
3.03	0.30
2.89	0.30
2.74	0.13
2.60	0.07
2.51	0.07
2.41	0.02
2.36	0.07
2.28	0.07
2.21	0.07
2.15	0.02
2.11	0.07
2.08	0.07
2.01	0.02
1.93	0.02
1.84	0.02

84

d(A)	I/I_1
14.5	0.20
12.6	0.20
9.31	0.07
8.00	0.27
7.20	0.27
6.24	1.00
5.74	0.42
5.39	0.50
4.98	0.37
4.82	0.75
4.61	0.37
4.22	0.27
4.01	0.15
3.78	0.20
3.57	0.20
3.40	0.37
3.06	0.20
2.89	0.15
2.78	0.20
2.48	0.10
2.39	0.10
2.18	0.15

85

d(A)	I/I_1
11.9	0.15
8.35	0.11
7.23	0.20
6.47	0.11
5.99	1.00
5.65	0.40
5.51	0.15
5.10	0.40
4.94	0.08
4.56	0.40
4.39	0.15
3.97	0.15
3.78	0.20
3.58	0.15
3.30	0.06
3.13	0.08
3.00	0.30
2.84	0.15
2.76	0.15
2.67	0.06
2.55	0.08
2.47	0.20
2.34	0.08
2.27	0.06
2.23	0.04
2.16	0.04
2.07	0.15
2.00	0.15
1.93	0.08
1.87	0.08
1.83	0.06

86

d(A)	I/I_1
11.19	0.50
8.55	0.20
7.14	0.27
6.28	0.10
5.79	1.00
5.50	0.27
4.65	0.50
4.28	0.20
4.08	0.20
3.87	0.15
3.72	0.15
3.58	0.15
3.45	0.10
3.32	0.10
3.24	0.15
3.14	0.15
3.06	0.15
2.92	0.09
2.81	0.10
2.70	0.10
2.60	0.09
2.40	0.15
2.21	0.09
2.15	0.09
2.08	0.10
2.01	0.04
1.92	0.04
1.83	0.02
1.76	0.01

87

d(A)	I/I_1
12.1	0.25
5.87	1.00
4.98	0.73
4.55	0.15
4.53	0.55
4.04	0.08
3.74	0.05
3.58	0.27
3.51	0.04
3.29	0.08
3.16	0.08
3.05	0.05
2.99	0.41
2.85	0.15
2.73	0.15
2.67	0.15
2.57	0.04
2.50	0.04
2.43	0.13
2.40	0.13
2.36	0.13
2.28	0.15
2.15	0.04
2.08	0.20
2.03	0.13
1.98	0.05
1.93	0.13
1.84	0.08

88

d(A)	I/I_1
11.9	0.30
9.45	0.20
7.40	0.30
6.51	0.40
6.22	0.40
5.97	1.00
5.47	0.73
5.14	0.53
4.85	0.40
4.69	0.73
4.27	0.73
4.03	0.02
3.74	0.73
3.50	0.07
3.37	0.20
3.24	0.02
3.10	0.20
2.98	0.13
2.89	0.13
2.82	0.02
2.76	0.13
2.67	0.13
2.55	0.07
2.50	0.03
2.44	0.40
2.30	0.40
2.24	0.13
2.23	0.02
2.16	0.13
2.11	0.13
2.06	0.07
1.98	0.13
1.93	0.02
1.89	0.02
1.85	0.02
1.81	0.07

89

d(A)	I/I_1
8.72	0.20
7.34	0.13
6.66	0.67
6.17	0.36
5.99	0.82
5.81	0.49
5.19	0.67
4.85	1.00
4.73	0.67
4.32	0.20
4.08	0.20
3.91	0.27
3.76	0.13
3.46	0.20
3.35	0.20
2.98	0.20
2.86	0.22
2.74	0.13
2.63	0.13
2.56	0.09
2.44	0.11
2.21	0.20
2.08	0.13
1.89	0.20

90

d(A)	I/I_1
11.7	0.06
11.4	0.08
6.54	0.08
5.91	1.00
5.45	0.20
4.90	0.30
4.54	0.20
4.19	0.06
4.10	0.20
3.92	0.06
3.63	0.20
3.48	0.04
3.36	0.04
3.26	0.06
3.12	0.11
3.04	0.01
2.84	0.06
2.66	0.11
2.52	0.11
2.46	0.04
2.34	0.11
2.31	0.11
2.22	0.03
2.10	0.08
2.01	0.03
1.96	0.08
1.91	0.01
1.86	0.08
1.81	0.01
1.75	0.01
1.64	0.06
1.54	0.06
1.39	0.03
1.25	0.01

91

d(A)	I/I_1
11.2	0.56
8.55	0.15
7.17	0.38
5.81	1.00
5.49	0.56
4.66	0.73
4.28	0.28
4.09	0.28
3.90	0.18
3.72	0.20
3.58	0.28
3.46	0.18
3.33	0.11
3.25	0.28
3.14	0.28
3.07	0.28
2.92	0.11
2.81	0.20
2.61	0.20
2.59	0.11
2.40	0.20
2.17	0.15
2.08	0.15
1.92	0.05
1.84	0.08

92

d(A)	I/I_1
11.4	0.13
8.23	0.13
7.23	0.27
6.23	0.27
5.89	0.04
5.59	0.67
5.33	1.00
5.11	0.27
4.90	0.27
4.62	0.27
4.50	0.02
4.37	0.27
4.20	0.36
3.98	0.02
3.81	0.09
3.60	0.36
3.38	0.01
3.24	0.20
3.15	0.20
3.00	0.20
2.93	0.20
2.81	0.20
2.74	0.20
2.65	0.01
2.57	0.20
2.48	0.20
2.43	0.20
2.35	0.20
2.30	0.01
2.26	0.20
2.21	0.20
2.16	0.02
2.01	0.13
1.98	0.01
1.84	0.01

93

d(A)	I/I_1
13.6	0.13
11.4	0.13
9.12	0.20
7.28	0.03
6.66	0.03
6.07	1.00
5.63	0.73
5.15	0.53
4.72	0.53
4.55	0.03
4.33	0.53
4.21	0.07
4.06	0.03
3.92	0.02
3.77	0.03
3.63	0.13
3.49	0.20
3.43	0.07
3.33	0.07
3.27	0.07
3.14	0.02
2.95	0.02
2.76	0.40
2.57	0.13
2.46	0.07
2.41	0.03
2.37	0.03
2.25	0.13
2.18	0.13
2.13	0.13
1.98	0.02
1.91	0.13
1.76	0.02

94

d(A)	I/I_1
13.1	0.15
6.89	0.10
6.56	0.20
6.24	0.15
5.85	1.00
5.70	0.75
5.50	0.15
5.22	0.15
4.93	0.10
4.80	0.27
4.42	0.37
4.25	0.07
4.18	0.10
4.09	0.01
3.96	0.01
3.85	0.07
3.75	0.07
3.64	0.20
3.47	0.07
3.33	0.27
3.28	0.01
3.18	0.01
3.12	0.15
3.06	0.01
2.98	0.15
2.92	0.07
2.83	0.20
2.74	0.01
2.67	0.01
2.62	0.10
2.58	0.10
2.53	0.01
2.47	0.03
2.44	0.01
2.40	0.10
2.35	0.10
2.31	0.03
2.27	0.02
2.21	0.10
2.15	0.07
2.09	0.07
2.05	0.03
1.98	0.03

95

d(A)	I/I_1
9.36	0.31
8.01	0.31
6.11	1.00
5.81	0.43
5.59	0.64
5.29	0.31
4.93	0.64
4.63	0.43
4.18	0.31
3.89	0.17
3.53	0.31
3.35	0.17
3.18	0.17
3.03	0.23
2.65	0.17
2.48	0.13
2.37	0.06
2.24	0.23

97

d(A)	I/I_1
13.0	0.03
10.6	0.25
8.89	0.25
7.94	0.13
6.63	0.13
6.22	1.00
5.83	0.25
5.31	0.75
4.97	0.25
4.71	0.56
4.34	0.56
4.00	0.13
3.72	0.13
3.64	0.13
3.38	0.03
3.16	0.03
2.94	0.25
2.85	0.13
2.65	0.03
2.50	0.03
2.34	0.03
2.26	0.03
2.19	0.13
2.11	0.13
2.07	0.13
1.98	0.13

96

11.1	0.27
8.31	0.36
7.23	0.49
6.24	0.36
5.91	0.09
5.59	0.49
5.32	1.00
5.10	0.13
4.87	0.13
4.68	0.27
4.52	0.13
4.39	0.36
4.21	0.01
3.82	0.09
3.60	0.36
3.24	0.13
3.13	0.09
3.04	0.20
2.94	0.20
2.84	0.13
2.75	0.09
2.71	0.04
2.66	0.04
2.62	0.27
2.55	0.27
2.47	0.20
2.34	0.27
2.30	0.09
2.24	0.02
2.19	0.09
2.15	0.27
2.11	0.04
2.06	0.13
2.01	0.13
1.98	0.01
1.93	0.13

98

d(A)	I/I_1
7.34	0.15
6.19	0.50
5.93	0.37
5.49	0.15
5.10	0.03
4.94	1.00
4.65	0.10
4.29	0.15
4.15	0.03
4.00	0.10
3.83	0.07
3.73	0.20
3.58	0.02
3.43	0.15
3.32	0.15
3.11	0.10
3.06	0.10
2.97	0.10
2.89	0.20
2.74	0.02
2.67	0.07
2.64	0.01
2.58	0.01
2.52	0.07
2.43	0.07
2.38	0.07
2.30	0.07
2.21	0.15
2.17	0.02
2.12	0.03
2.07	0.02
2.05	0.02
1.99	0.10
1.94	0.03
1.91	0.03
1.86	0.02

99

d(A)	I/I_1
14.7	0.10
10.4	0.27
7.50	0.20
6.84	0.15
6.15	0.27
5.37	1.00
5.07	0.27
4.46	0.37
4.30	0.20
3.79	0.20
3.58	0.20
3.40	0.20
3.25	0.15
3.09	0.20
2.93	0.01
2.84	0.01
2.65	0.10
2.57	0.03
2.49	0.07
2.40	0.01
2.31	0.01
2.26	0.10
2.21	0.10
2.16	0.10
2.08	0.20
1.97	0.10
1.89	0.07

100

d(A)	I/I_1
11.0	0.41
8.76	0.15
8.15	0.15
5.88	0.27
5.47	1.00
5.17	0.27
4.78	0.11
4.60	0.11
4.15	0.15
4.01	0.15
3.77	0.11
3.31	0.02
3.17	0.05
3.09	0.08
2.97	0.11
2.84	0.05
2.70	0.08
2.51	0.05
2.16	0.02
2.04	0.02

101

d(A)	I/I_1
14.7	0.27
9.66	0.02
7.69	0.27
6.56	0.10
5.96	1.00
5.64	0.15
5.22	0.01
4.95	0.50
4.70	0.27
4.40	0.02
4.13	0.15
3.87	0.20
3.74	0.20
3.60	0.20
3.44	0.15
3.28	0.20
3.16	0.01
3.04	0.15
2.89	0.10
2.75	0.01
2.65	0.01
2.57	0.15
2.47	0.10
2.34	0.10
2.28	0.15
2.19	0.15
2.14	0.10
2.04	0.03
1.94	0.03
1.85	0.01
1.63	0.01

102

d(A)	I/I_1
14.1	0.27
9.12	0.05
7.38	0.11
5.93	0.55
5.31	1.00
5.04	0.27
4.68	0.15
4.16	0.15
3.82	0.08
3.40	0.11
3.27	0.11
3.12	0.05
3.03	0.20
2.92	0.15
2.69	0.11
2.44	0.04
2.31	0.11
2.14	0.11
2.04	0.05
1.98	0.11

103

d(A)	I/I_1
14.4	0.28
6.01	1.00
5.77	0.56
5.56	0.75
5.35	0.38
5.13	1.00
4.85	0.75
4.71	0.38
4.50	0.28
3.83	0.28
3.72	0.47
3.55	0.15
3.41	0.38
3.31	0.11
3.08	0.38
2.94	0.47
2.64	0.11
2.56	0.15
2.38	0.20
2.28	0.20
2.21	0.20
2.13	0.15
2.06	0.15
2.00	0.15
1.92	0.15
1.83	0.08
1.75	0.08

104

d(A)	I/I_1
9.07	0.27
6.84	0.49
5.87	1.00
5.54	1.00
4.72	1.00
4.33	0.20
3.82	0.27
3.76	0.36
3.62	0.20
3.19	0.20
2.95	0.09
2.65	0.27
2.48	0.09
2.42	0.09
2.36	0.27
2.26	0.13
2.21	0.13

105

d(A)	I/I_1
14.6	0.20
10.9	0.13
9.21	0.53
7.63	0.40
6.26	0.13
5.83	1.00
5.59	0.40
5.29	0.40
4.91	0.53
4.51	0.53
4.27	0.30
4.11	0.40
3.95	0.07
3.73	0.40
3.60	0.07
3.41	0.40
3.25	0.30
3.13	0.30
3.04	0.20
2.86	0.07
2.76	0.07
2.70	0.13
2.61	0.13
2.55	0.07
2.47	0.30
2.40	0.30
2.34	0.30
2.22	0.30
2.15	0.13
2.10	0.13
2.06	0.30
1.99	0.13
1.88	0.13

106

d(A)	I/I_1
12.0	0.28
8.98	0.56
6.92	0.07
6.28	1.00
5.87	0.11
5.66	0.11
5.41	0.28
5.19	0.11
5.03	0.28
4.47	0.11
4.27	0.15
3.97	0.28
3.85	0.15
3.69	0.20
3.41	0.11
3.33	0.11
3.24	0.11
3.07	0.07
2.88	0.07
2.77	0.03
2.71	0.03
2.61	0.03
2.52	0.11
2.46	0.15
2.40	0.01
2.36	0.03
2.32	0.03
2.13	0.01
2.00	0.07
1.94	0.07
1.83	0.07

107

d(A)	I/I_1
12.8	0.04
9.82	0.27
7.20	0.55
6.61	0.20
5.89	1.00
5.44	0.41
5.17	0.41
4.89	0.86
4.59	0.41
4.31	0.27
4.10	0.20
3.91	0.20
3.76	0.55
3.56	0.20
3.35	0.02
3.20	0.20
3.09	0.15
2.96	0.20
2.85	0.11
2.71	0.11
2.62	0.15
2.45	0.18
2.29	0.20

108

d(A)	I/I_1
11.4	0.37
9.83	0.10
7.94	0.27
7.06	0.27
6.78	0.75
6.49	0.75
6.19	0.03
5.93	0.03
5.74	1.00
5.37	0.10
5.16	0.15
4.94	0.07
4.75	0.15
4.45	0.50
4.24	0.07
4.00	0.27
3.82	0.10
3.69	0.20
3.56	0.20
3.43	0.03
3.31	0.07
3.19	0.10
3.11	0.03
3.02	0.03
2.87	0.02
2.72	0.07
2.64	0.03
2.56	0.20
2.51	0.10
2.46	0.03
2.41	0.10
2.35	0.10
2.24	0.10
2.19	0.03
2.13	0.10
2.06	0.03
1.99	0.03

109

d(A)	I/I_1
12.9	0.27
8.63	0.03
6.22	1.00
5.87	0.50
5.42	0.50
5.13	0.37
4.87	0.07
4.72	0.75
3.54	0.20
3.25	0.03
3.09	0.02
2.88	0.03
2.45	0.05
2.35	0.07

111

d(A)	I/I_1
8.35	0.49
6.81	0.20
6.51	1.00
6.01	0.20
5.74	0.27
5.52	0.09
5.08	0.67
4.73	0.67
4.51	0.67
4.35	0.01
4.08	0.49
3.85	0.36
3.68	0.36
3.53	0.36
3.40	0.36
3.25	0.36
3.14	0.27
3.04	0.27
2.96	0.36
2.76	0.36
2.65	0.27
2.58	0.27
2.48	0.01
2.41	0.09
2.31	0.20
2.23	0.13
2.15	0.13
2.06	0.27
1.99	0.02
1.94	0.01
1.88	0.27
1.84	0.27
1.69	0.09
1.63	0.09

110

d(A)	I/I_1
15.1	0.27
12.9	0.10
11.9	0.03
10.3	0.27
9.61	0.20
7.83	0.02
6.86	0.02
6.39	0.15
5.93	0.27
5.66	1.00
5.26	0.37
4.96	0.37
4.76	0.15
4.62	0.20
4.47	0.27
4.25	0.27
4.15	0.37
3.97	0.15
3.84	0.03
3.63	0.07
3.45	0.15
3.32	0.07
3.16	0.10
3.03	0.07
2.95	0.07
2.67	0.02
2.65	0.03
2.57	0.03
2.46	0.03
2.41	0.03
2.35	0.07
2.29	0.03
2.23	0.07
2.13	0.07
2.07	0.07

112

d(A)	I/I_1
14.0	0.20
7.20	0.11
6.01	0.11
5.68	0.87
4.87	1.00
4.40	0.15
4.06	0.15
3.86	0.15
3.69	0.15
3.52	0.08
3.32	0.05
3.16	0.20
2.98	0.15
2.89	0.38
2.74	0.11
2.55	0.03
2.45	0.11
2.42	0.11
2.38	0.05
2.21	0.03
2.19	0.05
2.08	0.11
2.04	0.08
1.98	0.11
1.89	0.05
1.85	0.05
1.83	0.03

113

d(A)	I/I_1
13.3	0.05
11.0	0.27
7.56	1.00
7.05	0.27
6.24	0.73
5.81	0.18
5.47	0.27
5.19	0.18
4.91	0.27
4.78	0.86
4.60	0.73
4.42	0.37
4.10	0.18
3.94	0.55
3.78	0.55
3.61	0.18
3.48	0.18
3.38	0.27
3.24	0.09
3.16	0.18
3.07	0.09
2.92	0.18
2.82	0.05
2.73	0.27
2.65	0.05
2.61	0.05
2.55	0.05
2.50	0.05
2.43	0.27
2.31	0.27
2.25	0.05
2.21	0.27
2.15	0.05
2.08	0.09
2.04	0.18
1.95	0.05
1.85	0.05

114

d(A)	I/I_1
9.87	0.44
8.71	0.75
8.22	0.08
7.59	0.32
6.86	0.32
6.17	0.90
5.68	0.75
5.47	0.44
5.17	0.44
4.90	0.32
4.78	0.50
4.55	1.00
4.33	0.75
3.78	0.44
3.58	0.08
3.50	0.08
3.35	0.18
3.19	0.18
3.08	0.12
2.94	0.12
2.50	0.12
2.36	0.12
2.30	0.08
2.25	0.18
2.21	0.04
2.16	0.04
2.11	0.12

115

d(A)	I/I_1
12.1	0.09
9.61	0.13
8.84	0.13
8.04	0.49
7.20	0.36
6.68	0.09
6.07	0.43
5.45	1.00
4.89	0.83
4.51	0.09
4.32	0.43
4.03	0.13
3.85	0.20
3.59	0.27
3.40	0.20
3.23	0.24
2.52	0.13
2.45	0.09

116

d(A)	I/I_1
14.0	0.05
10.3	0.15
7.38	0.56
6.54	0.20
6.07	1.00
5.59	0.20
5.14	0.65
4.80	0.20
4.41	0.28
4.06	0.38
3.73	0.20
3.53	0.11
3.37	0.08
3.18	0.08
3.04	0.11
2.89	0.11
2.77	0.11
2.69	0.08
2.55	0.11
2.40	0.15
2.27	0.14
2.18	0.08
2.13	0.08
2.05	0.11

117

d(A)	I/I_1
14.5	0.06
7.31	0.53
6.20	1.00
5.65	1.00
5.25	0.30
5.10	0.37
4.72	0.33
4.50	0.13
4.27	0.23
4.09	0.15
3.88	0.15
3.72	0.30
3.52	0.17
3.34	0.20
3.12	0.11
3.01	0.06
2.85	0.11
2.72	0.03
2.64	0.15
2.57	0.01
2.47	0.08
2.34	0.03
2.28	0.04
2.21	0.04
2.05	0.11
2.02	0.11

118

d(A)	I/I_1
11.9	0.20
11.0	0.55
9.51	0.41
8.19	0.73
6.92	0.27
6.37	0.20
5.55	1.00
5.25	0.41
4.71	0.73
4.46	0.64
4.04	0.41
3.73	0.55
3.58	0.20
3.44	0.27
3.26	0.20
3.09	0.20
2.96	0.15
2.79	0.18
2.61	0.18
2.49	0.15
2.36	0.20
2.26	0.27
2.18	0.20
2.08	0.15
1.99	0.18
1.89	0.15

119

d(A)	I/I_1
12.4	0.15
10.9	0.07
9.66	0.01
8.76	0.75
6.92	0.37
6.64	1.00
6.01	0.20
5.49	0.20
5.29	0.50
5.15	0.50
4.60	0.27
4.08	0.20
3.94	0.37
3.73	0.15
3.62	0.15
3.50	0.27
3.33	0.07
3.16	0.20
3.03	0.03
2.88	0.15
2.74	0.07
2.65	0.07
2.54	0.10
2.40	0.07
2.31	0.07
2.17	0.10
2.10	0.07
2.01	0.15
1.98	0.15

120

d(A)	I/I_1
11.2	0.55
9.35	0.73
8.55	0.09
7.63	1.00
7.00	0.41
6.30	0.55
5.81	0.73
5.57	0.73
4.97	0.86
4.65	0.73
4.46	0.27
4.27	0.27
4.11	0.55
3.90	0.55
3.66	0.09
3.52	0.18
3.44	0.55
3.25	0.41
3.16	0.41
3.01	0.27
2.91	0.05
2.81	0.05
2.74	0.41
2.59	0.05
2.56	0.41
2.48	0.41
2.40	0.05
2.33	0.18
2.29	0.18
2.21	0.05
2.13	0.41
2.06	0.18
2.02	0.18
1.97	0.18
1.93	0.18
1.89	0.27
1.83	0.05
1.72	0.05

123

d(A)	I/I_1
14.0	0.20
11.7	0.01
7.73	0.15
8.63	0.02
7.66	0.15
7.14	0.10
6.15	1.00
5.72	0.27
5.04	0.27
4.21	0.15
3.82	0.20
3.60	0.15
3.35	0.10
3.05	0.10
2.56	0.03
2.45	0.02
2.11	0.01
2.00	0.01

121

d(A)	I/I_1
13.7	0.56
11.2	0.03
9.71	0.38
7.63	0.03
6.92	0.56
6.09	1.00
5.49	0.25
5.05	1.00
4.21	0.56
3.87	0.75
3.61	0.56
3.36	0.13
3.13	0.13

124

d(A)	I/I_1
14.0	0.13
9.41	0.13
7.05	0.13
6.11	0.73
5.72	1.00
5.44	0.03
5.21	0.40
4.72	0.40
4.38	0.40
3.58	0.30
3.19	0.30
3.05	0.20
2.82	0.13
2.64	0.03
2.53	0.07
2.38	0.03
2.31	0.07
2.14	0.03
2.05	0.07
1.97	0.03
1.86	0.03
1.71	0.03

122

d(A)	I/I_1
14.0	0.40
9.77	0.30
7.73	0.30
6.15	1.00
5.29	0.53
4.42	0.07
3.87	0.13
3.60	0.13
3.34	0.07
2.99	0.07

125

d(A)	I/I_1
15.0	0.20
11.1	0.03
8.23	0.11
7.20	0.03
5.99	0.11
5.54	1.00
5.01	0.11
4.73	0.11
4.42	0.20
4.24	0.08
4.00	0.08
3.77	0.11
3.62	0.08
3.42	0.06
3.10	0.20
2.99	0.06
2.81	0.04
2.72	0.11
2.52	0.08
2.47	0.06
2.40	0.04
2.34	0.08
2.30	0.08
2.23	0.08
2.16	0.04
2.09	0.08
2.03	0.04
1.92	0.01
1.86	0.03

126

d(A)	I/I_1
13.6	0.15
6.89	0.20
6.22	0.20
5.84	1.00
5.39	0.20
5.17	0.37
4.93	0.37
4.67	0.37
4.16	0.27
3.90	0.20
3.69	0.10
3.62	0.37
3.46	0.10
3.36	0.20
3.10	0.03
2.95	0.15
2.82	0.10
2.66	0.15
2.57	0.10
2.45	0.05
2.34	0.10
2.22	0.10
2.17	0.10
2.08	0.10
1.99	0.05
1.96	0.10

127

d(A)	I/I_1
14.7	0.07
13.1	0.15
11.8	0.03
10.8	0.20
9.41	0.03
7.97	0.10
7.28	0.20
7.02	0.75
6.51	0.50
6.15	1.00
5.70	0.67
5.33	0.75
5.02	0.92
4.81	0.50
4.67	0.50
4.41	0.10
4.30	0.15
4.15	0.03
3.75	0.27
3.51	0.15
3.19	0.10
3.02	0.10
2.76	0.02
2.61	0.03
2.47	0.02
2.34	0.03

128

d(A)	I/I_1
15.2	0.13
14.0	0.04
9.07	0.09
7.90	0.17
7.03	0.20
6.21	1.00
5.87	0.13
5.52	1.00
4.94	0.13
4.71	0.01
4.38	0.13
4.17	0.83
3.92	0.20
3.72	0.27
3.50	0.27
3.34	0.09
3.25	0.09
3.08	0.13
2.97	0.09
2.85	0.04
2.77	0.13
2.68	0.02
2.56	0.01
2.46	0.09
2.37	0.20
2.29	0.09
2.23	0.04
2.17	0.02
2.11	0.01
2.07	0.09
1.97	0.02
1.93	0.02
1.85	0.02
1.80	0.02
1.75	0.02

129

d(A)	I/I_1
15.4	0.20
10.7	0.15
9.67	0.15
7.90	0.15
7.38	0.11
6.19	0.75
6.07	0.28
5.93	1.00
5.49	0.28
5.39	0.75
4.82	0.38
4.26	0.38
3.85	0.15
3.52	0.28
3.36	0.11
3.20	0.11
2.90	0.08
2.72	0.20
2.57	0.20
2.14	0.11
2.07	0.11
2.04	0.11

130

d(A)	I/I_1
15.8	0.11
9.26	0.30
7.22	0.30
6.84	0.20
5.87	1.00
5.59	0.20
5.31	1.00
5.08	0.30
4.64	0.40
4.29	0.20
3.96	0.20
3.56	0.30
3.30	0.11
3.11	0.15
2.88	0.15
2.75	0.15
2.60	0.15
2.58	0.08
2.36	0.06
2.31	0.08
2.17	0.06
2.08	0.08
2.00	0.15
1.93	0.04
1.88	0.06
1.82	0.01
1.76	0.04

131		132		133	
d(A)	I/I_1	d(A)	I/I_1	d(A)	I/I_1
15.0	0.07	12.0	0.13	13.7	0.20
9.07	0.20	10.8	0.20	9.41	0.09
8.01	0.07	8.98	0.13	7.06	0.36
7.28	0.30	7.17	0.36	6.49	1.00
7.00	0.30	6.09	0.67	5.79	0.67
6.26	0.40	5.63	0.09	5.47	0.49
5.95	1.00	5.29	1.00	5.26	0.01
5.63	0.53	5.11	1.00	5.05	1.00
5.32	0.13	4.66	0.13	4.81	0.01
5.00	0.40	4.43	0.36	4.63	0.36
4.80	0.40	4.17	0.27	4.34	0.36
4.62	0.40	3.98	0.27	4.16	0.36
4.32	0.73	3.78	0.13	3.96	0.36
4.10	0.30	3.59	0.09	3.78	0.36
3.93	0.30	3.47	0.20	3.63	0.36
3.75	0.30	3.32	0.02	3.40	0.36
3.62	0.07	3.21	0.27	3.26	0.36
3.52	0.07	3.08	0.20	3.19	0.36
3.39	0.07	2.96	0.13	3.08	0.20
3.30	0.20	2.91	0.13	2.95	0.13
3.23	0.07	2.74	0.04	2.87	0.13
3.06	0.07	2.59	0.20	2.75	0.36
2.89	0.07	2.46	0.20	2.69	0.04
2.75	0.07	2.31	0.13	2.65	0.04
2.60	0.03	2.23	0.13	2.54	0.09
2.52	0.02	2.15	0.13	2.48	0.01
2.43	0.03	2.09	0.13	2.41	0.09
2.37	0.02	2.05	0.13	2.32	0.09
2.20	0.02	1.99	0.09	2.27	0.01
2.10	0.02	1.94	0.09	2.22	0.01
2.05	0.02	1.90	0.13	2.18	0.27
2.01	0.02	1.83	0.09	2.10	0.01
1.95	0.02	1.78	0.02	2.03	0.27
		1.74	0.09	2.02	0.01
		1.70	0.01	1.97	0.01
		1.64	0.01	1.90	0.01
				1.86	0.09

134

d(A)	I/I_1
11.3	0.15
9.02	0.15
7.53	0.38
6.71	0.28
6.42	0.75
5.75	0.11
5.26	1.00
4.73	0.11
4.51	0.08
4.24	0.56
4.04	0.32
3.92	0.32
3.81	0.28
3.63	0.32
3.60	0.28
3.47	0.32
3.33	0.11
3.03	0.20
2.92	0.11
2.79	0.08
2.65	0.11
2.59	0.05
2.53	0.08
2.37	0.15
2.33	0.05
2.24	0.05
2.13	0.11
2.08	0.03
1.99	0.08

135

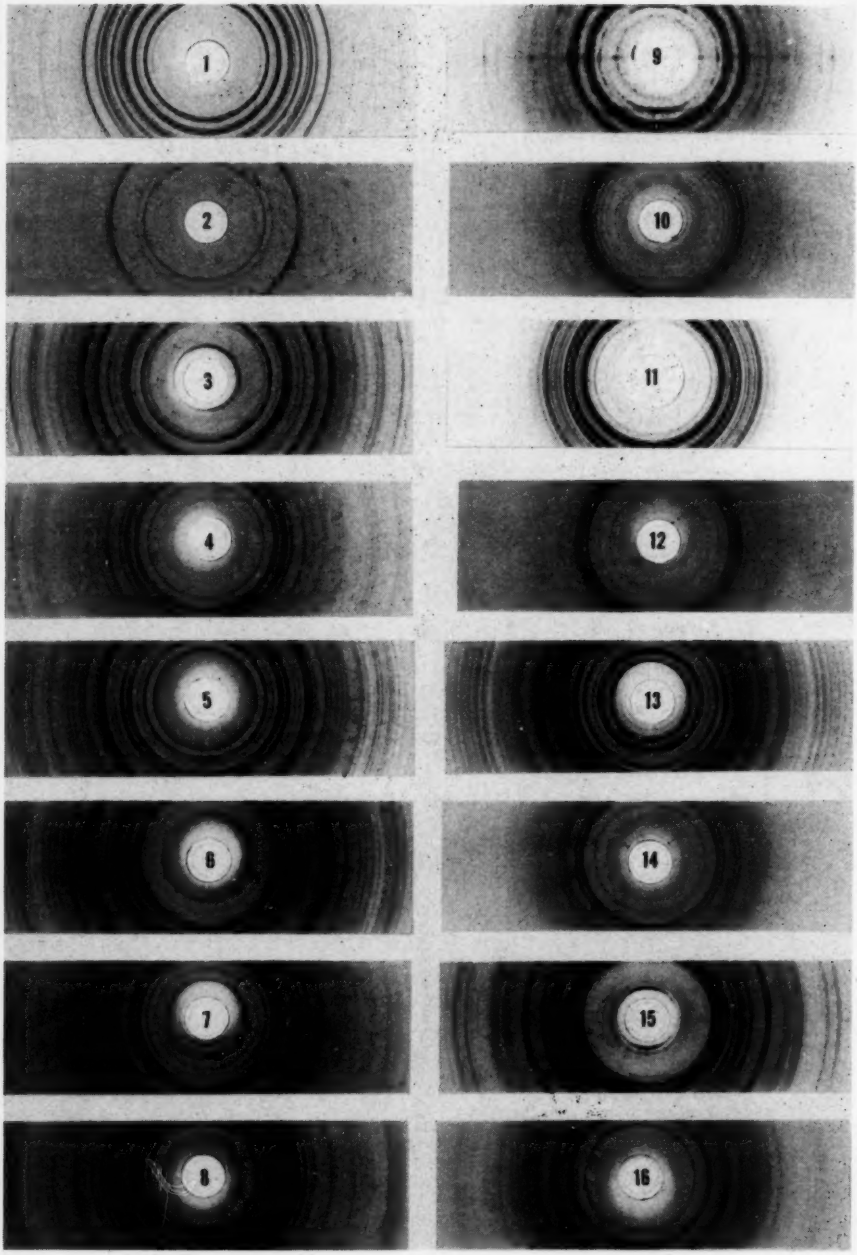
d(A)	I/I_1
9.72	0.47
7.22	0.15
6.49	0.75
5.88	0.56
5.57	0.75
5.31	0.08
5.10	1.00
4.90	0.38
4.47	0.38
4.20	0.47
3.88	0.15
3.73	0.38
3.56	0.28
3.41	0.28
3.23	0.11
3.14	0.33
2.97	0.15
2.88	0.05
2.80	0.05
2.70	0.15
2.65	0.05
2.54	0.05
2.50	0.15
2.44	0.08
2.34	0.11
2.28	0.15
2.17	0.15
2.12	0.03
2.07	0.05
2.03	0.05

136

d(A)	I/I_1
13.5	0.75
10.8	0.15
7.83	0.20
6.86	0.37
6.46	0.50
6.11	0.07
5.64	0.27
5.14	1.00
4.87	0.50
4.44	0.15
4.29	0.50
4.07	0.15
3.89	0.10
3.71	0.15
3.51	0.27
3.43	0.20
3.30	0.23
3.13	0.27
2.65	0.20
2.42	0.07
2.24	0.07

137

d(A)	I/I_1
9.41	0.15
8.23	0.11
7.47	0.27
5.85	0.55
5.26	0.02
4.85	1.00
4.45	0.73
4.21	0.20
3.95	0.27
3.74	0.11
3.55	0.34
3.42	0.41
3.16	0.04
3.06	0.04
2.91	0.24
2.56	0.11
2.51	0.15
2.40	0.05
2.31	0.05
2.25	0.02
2.19	0.15
2.04	0.02
1.95	0.04
1.88	0.05
1.84	0.08
1.76	0.08
1.68	0.02
1.65	0.02
1.60	0.02
1.56	0.04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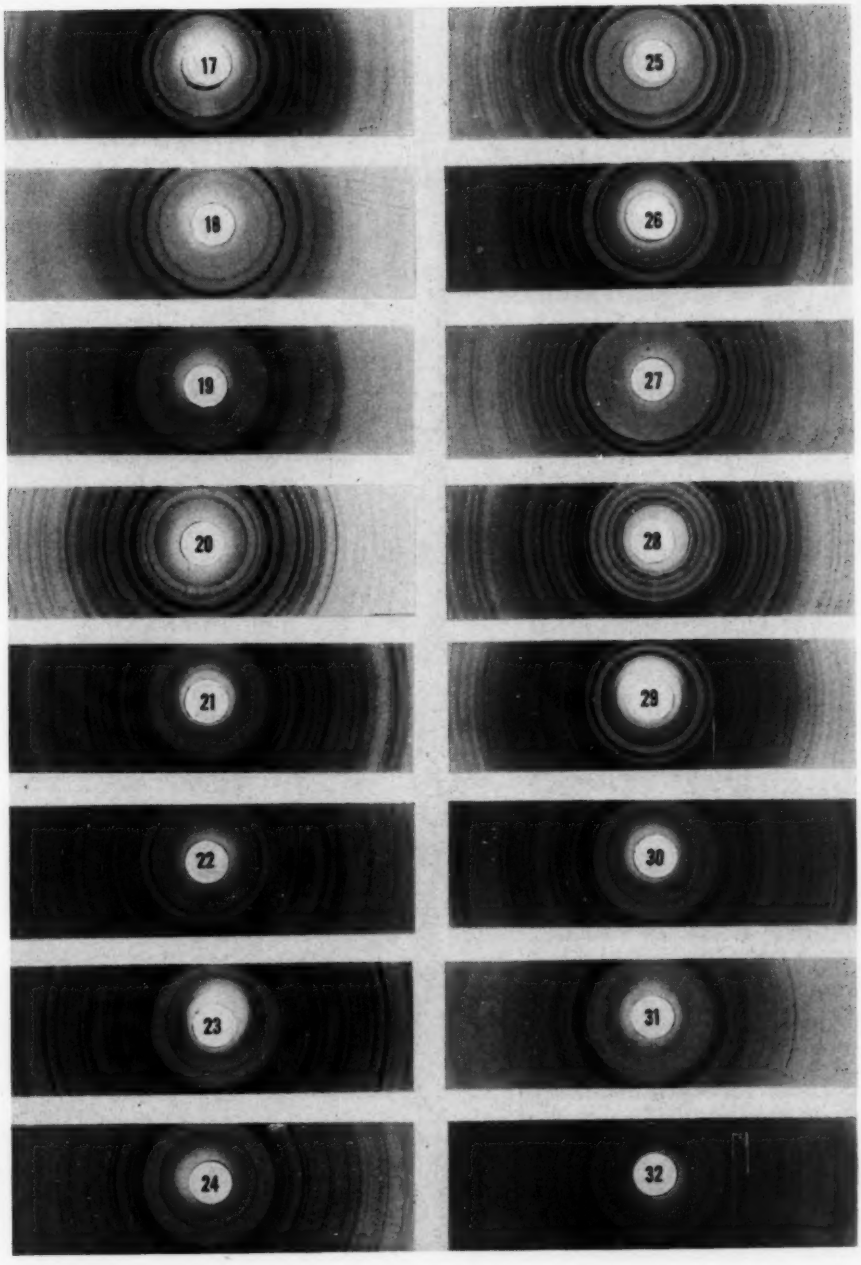
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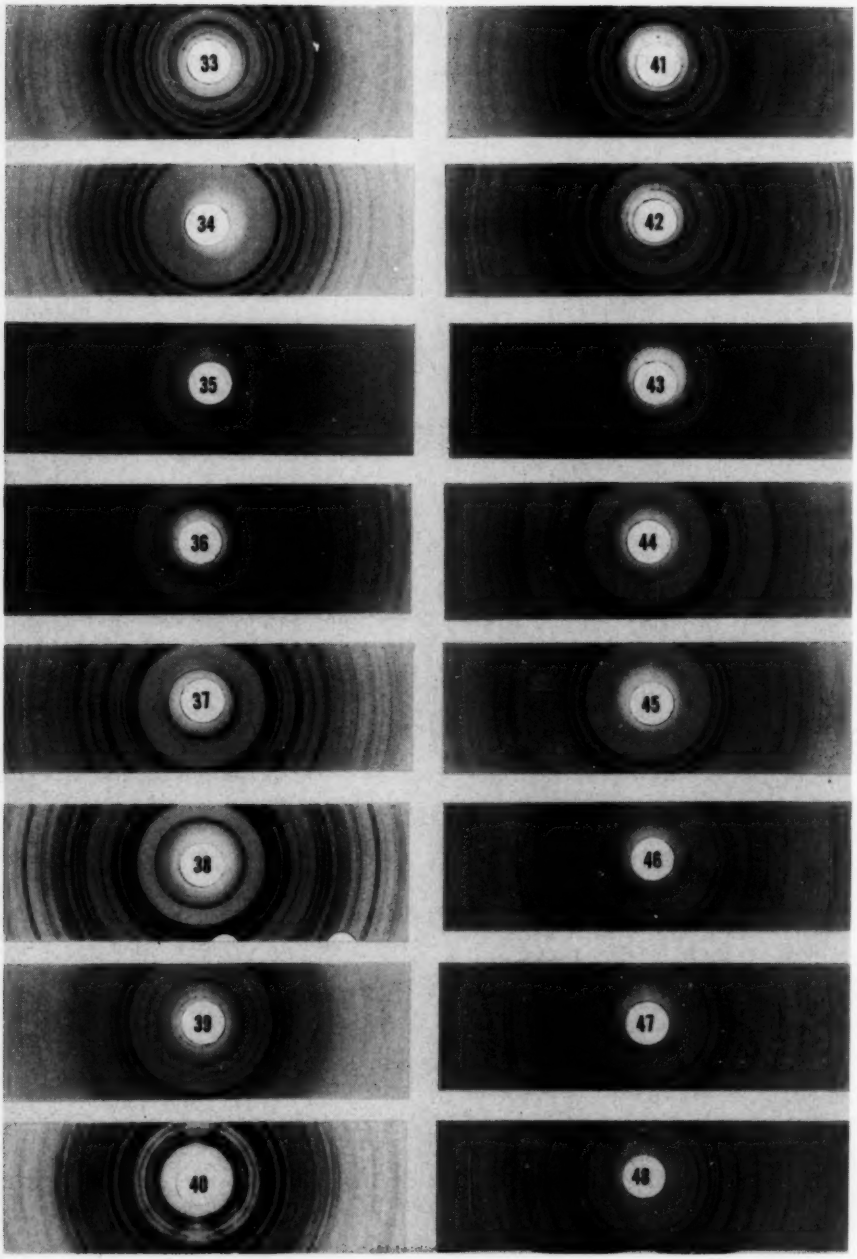
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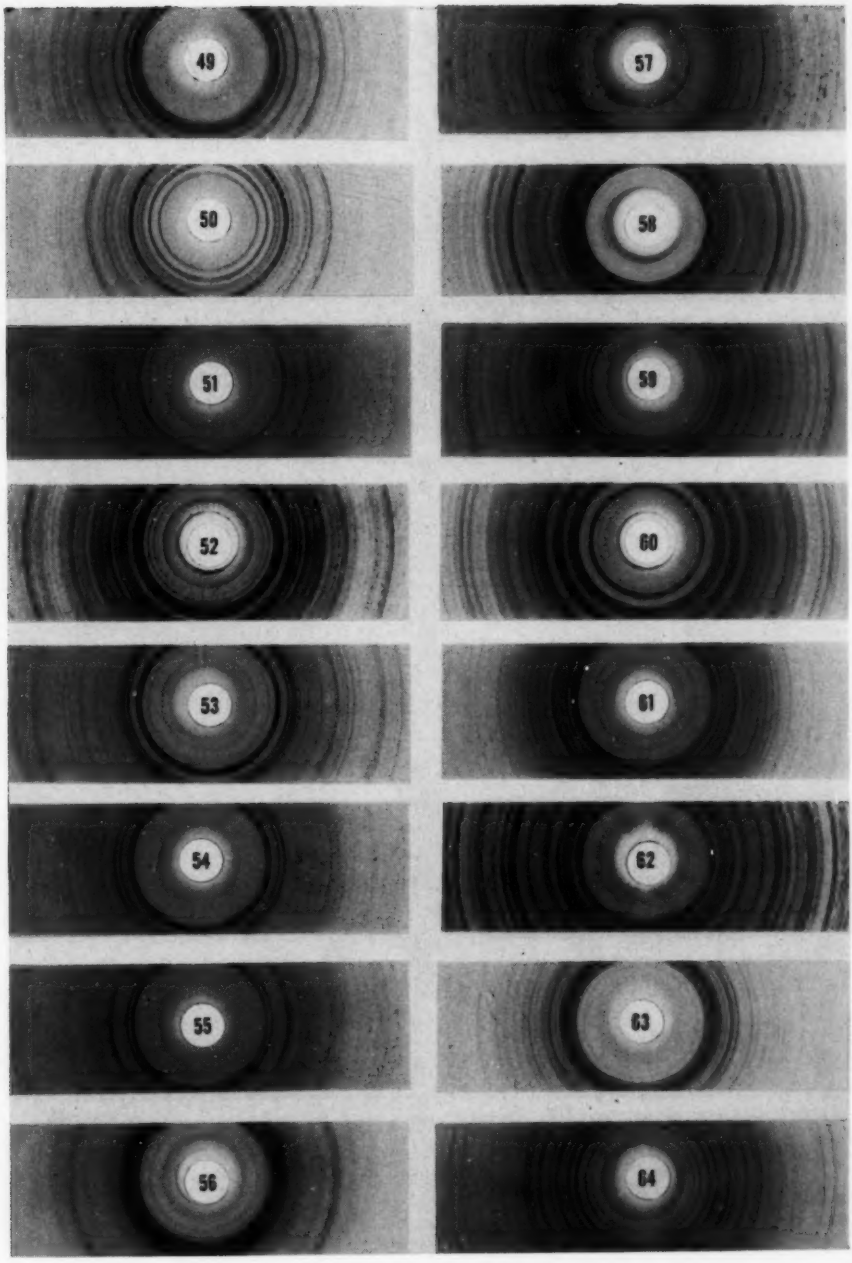
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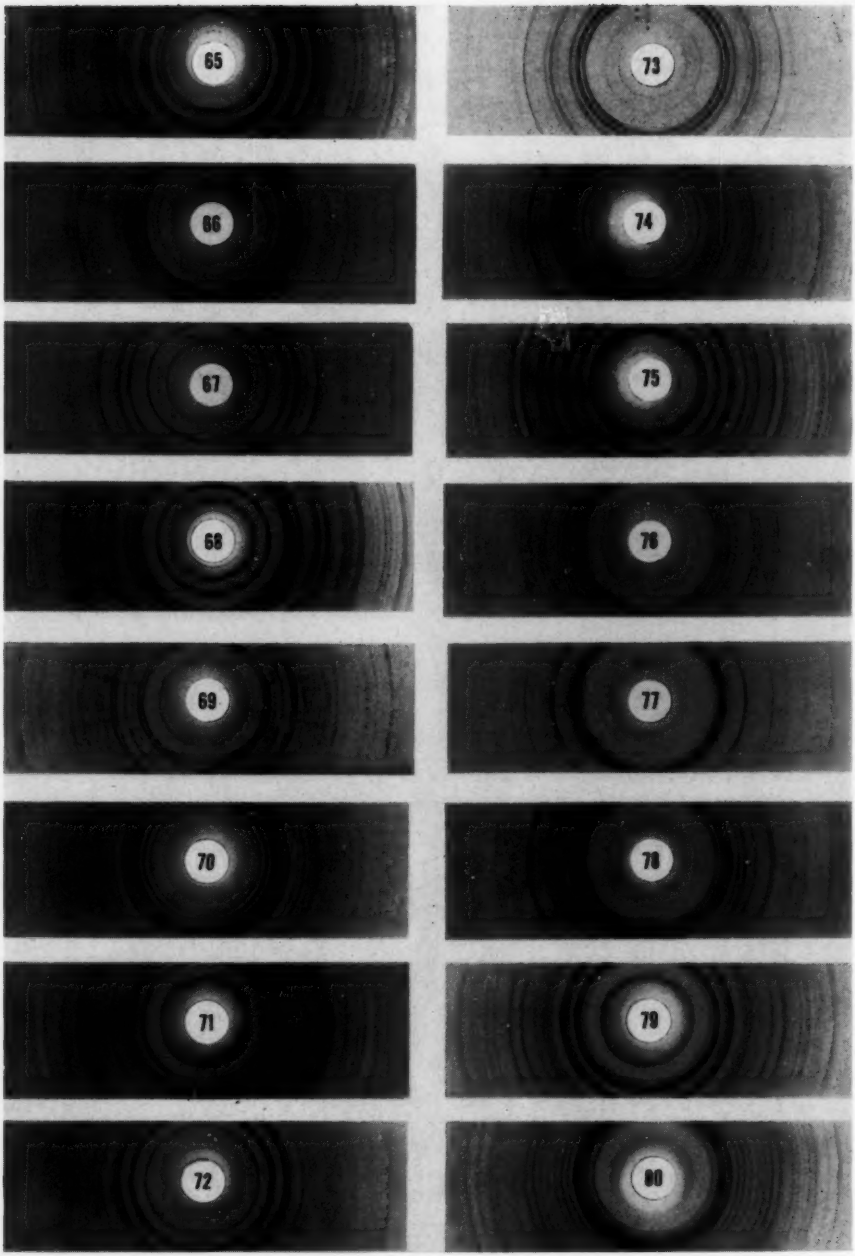
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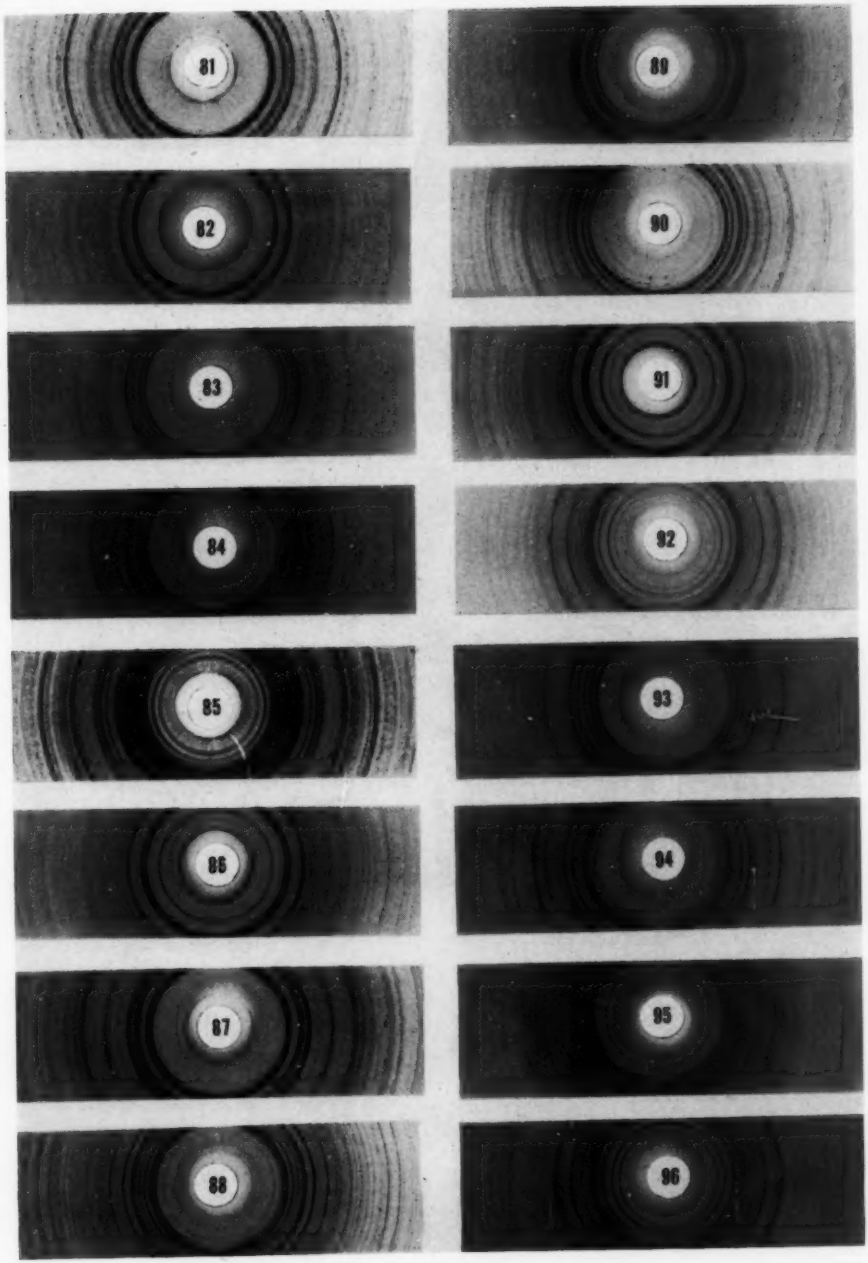
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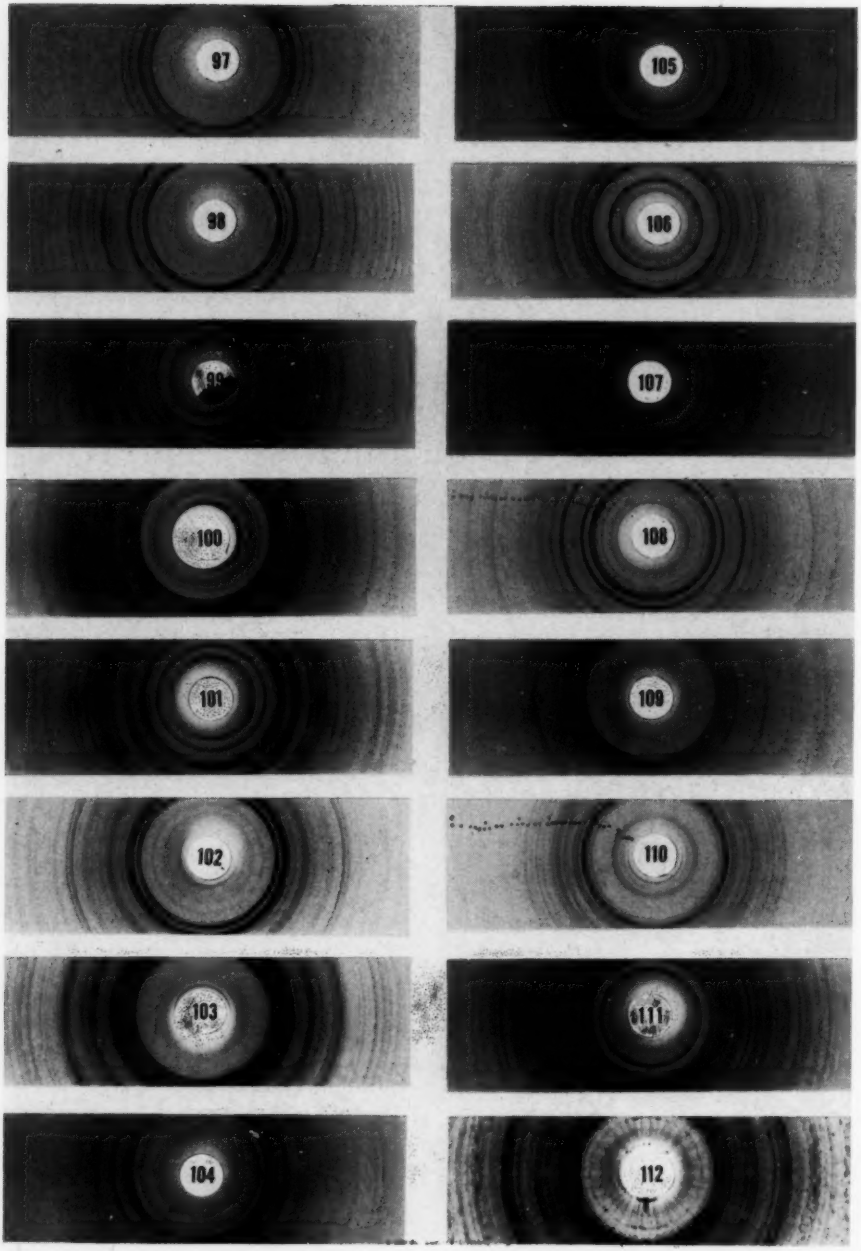
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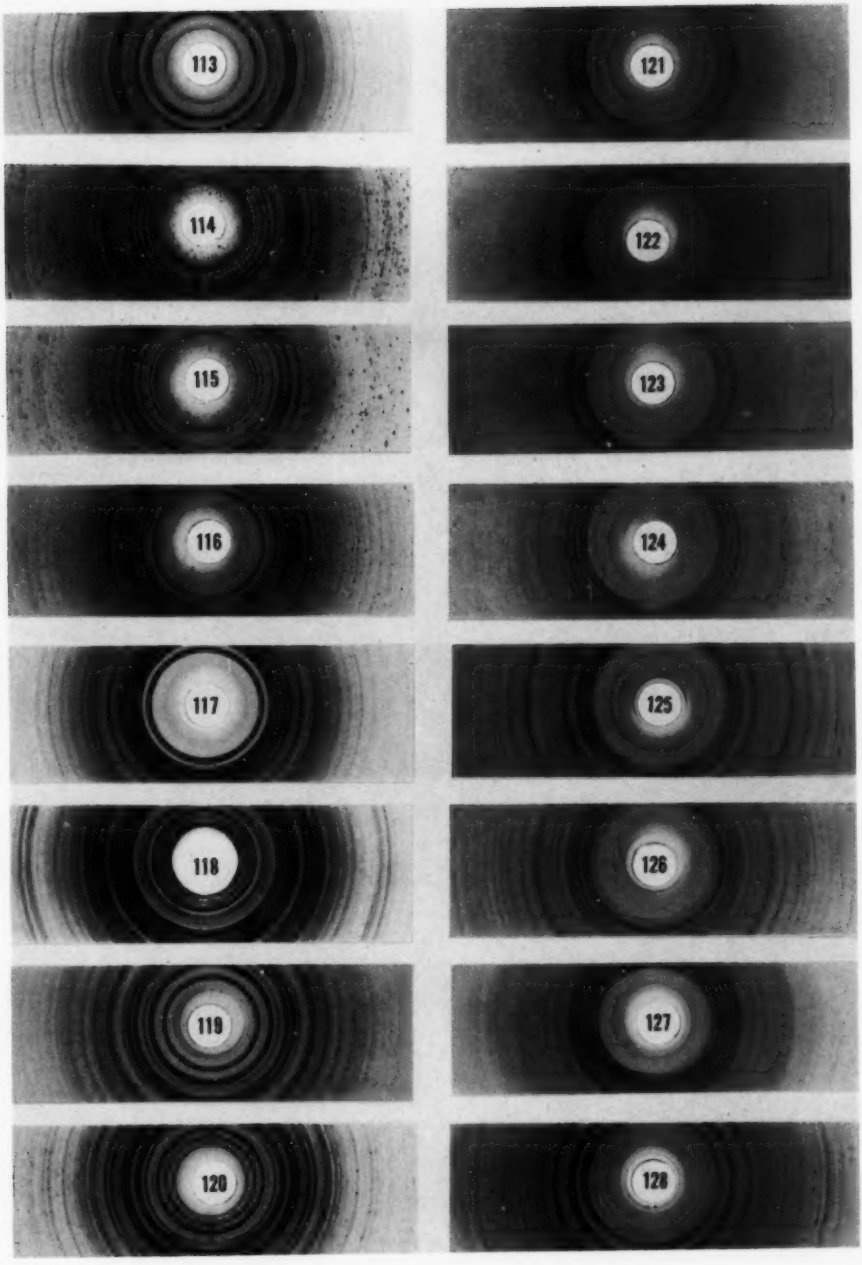
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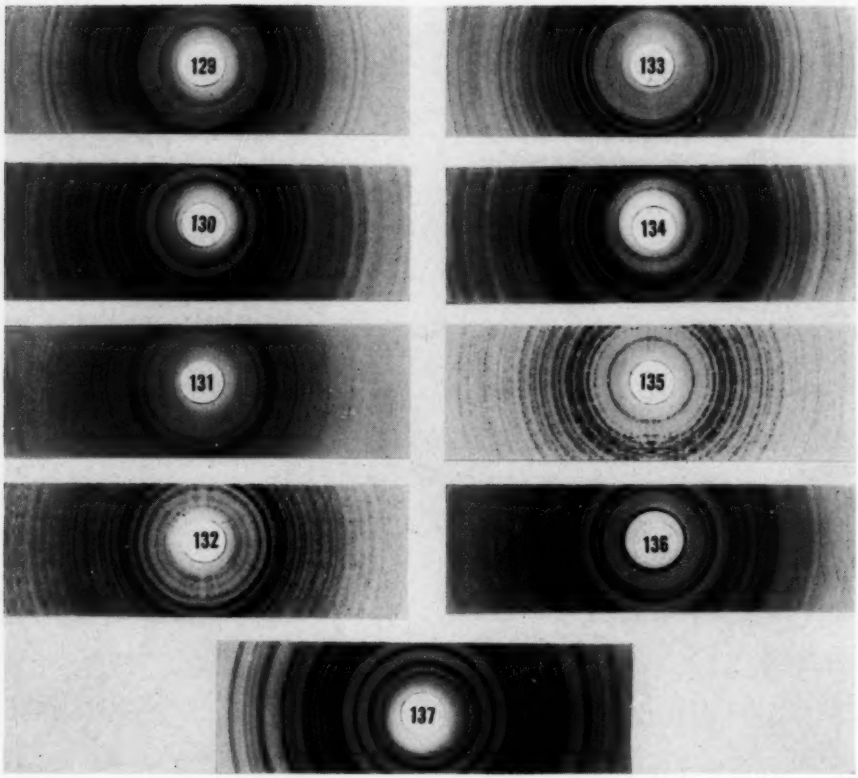
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